In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Filed: April 11, 2025

MEGHAN TRUE,	*	PUBLISHED
Petitioner,	*	No. 21-2110V
v.	*	Special Master Nora Beth Dorsey
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	Dismissal Decision; Human Papillomavirus ("HPV") Vaccine; Transverse Myelitis ("TM").
Respondent.	*	

Laura Levenberg, Muller Brazil PA, Dresher, PA, for Petitioner.
Felicia Langel, U.S. Department of Justice, Washington, DC, for Respondent.

DECISION[1]

On October 29, 2021, Meghan True[2] ("Petitioner") filed a petition for compensation under the National Vaccine Injury Compensation Program ("Vaccine Act" or "the Program"), 42 U.S.C. § 300aa-10 et seq. (2018),[3] alleging that as a result of a third human papillomavirus ("HPV") vaccine administered on June 7, 2019, she developed transverse myelitis ("TM"). Petition at Preamble (ECF No. 1). Respondent argued against compensation, stating

[1] Because this Decision contains a reasoned explanation for the action in this case, the undersigned is required to post it on the United States Court of Federal Claims' website and/or at https://www.govinfo.gov/app/collection/uscourts/national/cofc in accordance with the E-Government Act of 2002. 44 U.S.C. § 3501 note (2018) (Federal Management and Promotion of Electronic Government Services). **This means the Decision will be available to anyone with access to the Internet.** In accordance with Vaccine Rule 18(b), Petitioner has 14 days to identify and move to redact medical or other information, the disclosure of which would constitute an unwarranted invasion of privacy. If, upon review, the undersigned agrees that the identified material fits within this definition, the undersigned will redact such material from public access.

[2] During pendency of this matter, Petitioner legally changed her name.

[3] The National Vaccine Injury Compensation Program is set forth in Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755, codified as amended, 42 U.S.C. §§ 300aa-10 to -34 (2018) ("Vaccine Act" or "the Act"). All citations in this Decision to individual sections of the Vaccine Act are to 42 U.S.C.A. § 300aa.

"[P]etitioner has not met her burden of establishing entitlement to compensation under the terms of the Vaccine Act." Respondent's Report ("Resp. Rept.") at 1 (ECF No. 23).

After carefully analyzing and weighing the evidence presented in accordance with the applicable legal standards,[4] the undersigned finds Petitioner failed to provide preponderant evidence that her HPV vaccination caused her to develop TM. Thus, Petitioner has failed to satisfy her burden of proof under Althen v. Secretary of Health & Human Services, 418 F.3d 1274, 1280 (Fed. Cir. 2005). Accordingly, the petition must be dismissed.

I. ISSUES TO BE DECIDED

The parties stipulated that Petitioner received her third HPV vaccination on June 7, 2019 and that this vaccine is on the Vaccine Injury Table and was administered in the United States. Joint Submission, filed May 28, 2024, at 1 (ECF No. 59).

At dispute is "[t]he nature and diagnosis of [P]etitioner's alleged injuries." Joint Submission at 1. Petitioner contends her post-vaccination diagnosis is TM, while Respondent disagrees. Petitioner's Motion for Ruling on the Record ("Pet. Mot."), filed May 28, 2024, at 11-12 (ECF No. 57); Resp. Response to Pet. Mot. ("Resp. Response"), filed July 29, 2024, at 11-13 (ECF No. 60); Pet. Reply to Resp. Response ("Pet. Reply"), filed Aug. 21, 2024, at 1 (ECF No. 63). Respondent's experts contend Petitioner's symptoms were most consistent with fibromyalgia and/or a functional neurological disorder ("FND"). Resp. Exhibits ("Exs.") A-C.

The parties also dispute "[w]hether the HPV vaccine administered to [P]etitioner on June 7, 2019[] caused her alleged injuries." Joint Submission at 1. The parties agree all three Althen prongs remain in dispute. Pet. Mot. at 6-17; Resp. Response at 10-18; Pet. Reply at 2-4.

II. BACKGROUND

A. Procedural History

Petitioner filed her petition on October 29, 2021, followed by medical records and an affidavit.[5] Petition; Pet. Exs. 1-10. On March 25, 2022, this case was assigned to the undersigned. Notice of Reassignment dated Mar. 25, 2022 (ECF No. 18). On June 20, 2022, Respondent filed his Rule 4(c) report, arguing against compensation. Resp. Rept. at 1.

[4] While the undersigned has reviewed all of the information filed in this case, only those filings and records that are most relevant will be discussed. See Moriarty v. Sec'y of Health & Hum. Servs., 844 F.3d 1322, 1328 (Fed. Cir. 2016) ("We generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision."); see also Paterek v. Sec'y of Health & Hum. Servs., 527 F. App'x 875, 884 (Fed. Cir. 2013) ("Finding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered.").

[5] Medical records were filed throughout litigation.

From November 2022 to October 2023, Petitioner filed expert reports from Dr. Lawrence Steinman and Respondent filed expert reports from Dr. Dara G. Jamieson and Dr. Mehrdad Matloubian. Pet. Exs. 11, 32; Resp. Exs. A-C.

The parties requested a Rule 5 conference, which occurred December 14, 2023. Rule 5 Order dated Dec. 14, 2023 (ECF No. 51). The undersigned preliminarily found Petitioner's post-vaccination diagnosis was TM. Id. at 2. She was unable to provide any preliminary findings as to causation. Id. at 3.

Thereafter, Respondent indicated he wished to continue to defend this case, and the parties agreed to resolve the question of entitlement through a ruling on the record. Joint Status Rept., filed Feb. 28, 2024 (ECF No. 53); Joint Status Rept., filed Mar. 29, 2024 (ECF No. 55). Petitioner filed her motion for a ruling on the record on May 28, 2024. Pet. Mot. Respondent filed his responsive brief on July 29, 2024, and Petitioner filed a reply on August 21, 2024. Resp. Response; Pet. Reply.

This matter is now ripe for adjudication.

B. Summary of Relevant Medical Records[6]

Petitioner's prior medical history was significant for chronic left knee pain and stiffness that extended to the ankle, monthly migraines, anxiety, and depression. See generally Pet. Ex. 3; Pet. Ex. 2 at 14-15. Petitioner had no neurological issues in the three years prior to vaccination. See Pet. Mot. at 2; Resp. Response at 2.

On September 26, 2018, Petitioner visited her primary care provider ("PCP"). Pet. Ex. 2 at 14. Petitioner complained of pain in her left knee, monthly migraines, and periodic chest pain and shortness of breath. Id. at 16. Examination showed Petitioner was hyperreflexic.[7] Id. at 17. Petitioner received her first of three HPV vaccinations at this visit. Id.; Pet. Ex. 1 at 4. No adverse reaction was noted. See Pet. Ex. 2 at 14-17.

Petitioner returned to her PCP on November 21, 2018 for her second HPV vaccination. Pet. Ex. 2 at 12-13; Pet. Ex. 1 at 4. No complaints were noted. Pet. Ex. 2 at 12-13.

[6] This summary of medical records is largely taken from the parties' briefs, with additions and edits from the undersigned, as the undersigned finds they provided an accurate representation of the records. See Pet. Mot. at 2-4; Resp. Response at 2-7.

[7] Hyperreflexia is the "exaggeration of reflexes." Hyperreflexia, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=23992 (last visited Mar. 18, 2025).

Petitioner received her third and final HPV vaccination from her PCP on June 7, 2019,[8] at 23 years of age. Pet. Ex. 2 at 10; Pet. Ex. 1 at 4.

On June 10, 2019, three days post-vaccination, Petitioner presented to the emergency department ("ED") complaining of leg weakness and fatigue. Pet. Ex. 4 at 311. Petitioner reported that three days prior (June 7), she received her third HPV vaccination "and felt itchy and fatigue shortly thereafter." Id. She reported she had similar symptoms after the first two HPV vaccinations that resolved within a couple of days. Id. at 311-12. However, her symptoms began to worsen two days prior (June 8). Id. at 312. Then, the day before (June 9), "she began to experience extremity weakness." Id. Petitioner reported occasional leg spasms and falls, with two falls the day before (June 9) and multiple falls at work prior to presenting to the ED on June 10. Id. She also complained of shortness of breath, back pain, neck pain, and headache. Id.

ED physician Eric Ruschman, M.D., noted on examination that Petitioner was "profoundly weak," with difficulty standing and ambulating. Pet. Ex. 4 at 313. Dr. Ruschman noted that "despite great efforts to get [Petitioner] up to walk she really is incredibly weak in her legs and cannot make a step without any assistance and even this is difficult." Id. at 314. Petitioner also had bilateral knee hyperreflexia with 3+ knee jerks and 2+ ankle jerks. Id. at 313. Dr. Ruschman wrote, "This is a young lady without any significant past medical history who recently had her third HPV vaccine. However she was feeling fatigued even before this and whether that has anything to do with her sudden neurologic deterioration is unclear." Id. at 314. Dr. Ruschman ruled out Guillain-Barré Syndrome ("GBS") due to Petitioner's hyperreflexia but he noted that "[s]he could have something like [TM[9]] or abrupt onset of multiple sclerosis [("MS")] or other demyelinating disease." Id. Dr. Ruschman conferred with the on-call neurologist and they agreed to admit Petitioner for diagnostic testing and three days of Solu-Medrol. Id. While in the ED, Petitioner received her first Solu-Medrol dose. Id. at 319.

Wyckiffe Opii, M.D., conducted a history and physical examination of Petitioner that evening (June 10). Pet. Ex. 4 at 315. History of present illness documented that Petitioner presented to the ED for "[three] days of progressively worsening lower extremity weakness." Id. Petitioner reported

[8] There are no records from Petitioner's PCP documenting Petitioner's visit on June 7, 2019. However, there are records indicating Petitioner's third HPV vaccination was administered on this date. See Pet. Ex. 2 at 10 (documenting third HPV vaccination was administered on June 7, 2019 during a visit to the PCP on June 20, 2019); see also Pet. Ex. 1 at 4. And the parties stipulated that this vaccine was administered on June 7, 2019. Joint Submission at 1.

[9] TM, a central nervous system ("CNS") disorder, "is a focal inflammatory disorder of the spinal cord, resulting in motor, sensory, and autonomic dysfunction." Resp. Ex. A-3 at 1 (Transverse Myelitis Consortium Working Group, Proposed Diagnostic Criteria and Nosology of Acute Transverse Myelitis, 59 Neurology 499 (2002)); see also Pet. Ex. 34 at 6-7 (Benjamin Greenberg, Transverse Myelitis, UpToDate, https://www.uptodate.com/contents/transverse-myelitis (last updated Feb. 14, 2023)) (an older version, last updated April 25, 2022, was filed as Resp. Ex. B-3).

> her symptoms began this past Friday,[10] with generalized fatigue, come Saturday[11] she did have generalized weakness[,] difficulty walking, [and] had [two] falls. She mentioned that she woke up on Sunday[12] feeling a little bit better but, that later in the afternoon symptoms recurred. She went to work today, [June 10,] was unable to ambulate, fell[,] and was transported to the ED.

Id. At evaluation, "[Petitioner] mentioned that she received the third dose of [HPV] vaccine on Friday," June 7. Id. Physical examination revealed "generalized bilateral lower extremity weakness 3/5." Id. at 317. Dr. Opii's assessment stated Petitioner "present[ed] to the ED with [three] days of progressively worsening lower extremity weakness. Admitted with suspicion for likely highly MS vs suspected [TM]." Id. at 318.

The next morning, on June 11, 2019, Petitioner had a consultation with neurologist Stephanie D. Sheffield, M.D. Pet. Ex. 4 at 319. Dr. Sheffield noted Petitioner received her third HPV vaccination which was followed by "her typical fatigue and malaise [] except fatigue was worse than usual." Id. Then, two days prior, on June 9, Petitioner noticed "her legs felt heavy," followed by multiple falls due to progressive weakness. Id. Petitioner reported "a stiff tight sensation in her legs," but otherwise no other sensory changes. Id. She "had episodes of trembling or spasming [in her legs] that seem[] to be brought on by tactile stimulus." Id. Petitioner also reported new neck and back pain, but had no weakness of her arms or difficulty with bladder or bowel control, speech, or swallowing. Id. Dr. Sheffield noted magnetic resonance imaging ("MRI") of Petitioner's brain, cervical spine, thoracic spine, and lumbar spine with and without contrast were conducted and were unremarkable other than a minimal disc bulge at L4-L5. Id. at 319, 326-27. Physical examination revealed mild weakness in lower extremities (hip flexion, knee flexion, knee extension, and foot dorsiflexion), hyperreflexia, diminished pinprick sensation in lower extremities at the L1-L2 spinal level, and spasticity in lower extremities. Id. at 320. Assessment was "[m]yelopathy—likely [TM] with negative scan, [o]nset post vaccination." Id. at 321. Dr. Sheffield noted "[p]ossible improvement already this morning after [one] dose of Solu-Medrol with no spasms triggered by tactile stimulation." Id. Lumbar puncture was ordered and showed "nothing alarming."[13] Id. at 321, 324-25, 345.

By the afternoon of June 11, Petitioner was "feeling better" and was able to walk to the bathroom. Pet. Ex. 4 at 339. Hospitalist Kathryn Woody, M.D., evaluated Petitioner and noted Petitioner was "admitted with progressive bilateral leg weakness presumed to be [TM]." Id. at 342. Assessment was "[p]resumed [a]cute [TM]." Id.

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[10] Friday was June 7, 2019, the date of vaccination.

[11] Saturday was June 8, 2019, one day after vaccination.

[12] Sunday was June 9, 2019, two days post-vaccination.

[13] Cerebrospinal fluid ("CSF") results showed elevated glucose (92; reference range: 40-70); normal protein (20.5; reference range: 15.0-45.0), white blood cells (1, 3; reference range: 0-5), and red blood cells (0, 1; reference range: 0-5); and negative PCR meningitis/encephalitis panel and MS profile. Pet. Ex. 4 at 344, 346, 387-93.

On the morning of June 12, 2019, Petitioner reported her lower extremities felt stronger and she had normal sensation. Pet. Ex. 4 at 332. Petitioner demonstrated a "slow and guarded gait with no reciprocal arm swing." Id. at 333. Later that morning, Dr. Sheffield saw Petitioner who reported she was feeling better and no longer had stiffness in her legs. Id. at 346. Dr. Sheffield noted Petitioner was "markedly improved with Solu-Medrol." Id. Assessment was TM. Id. She noted "MS [was] not ruled out" although it was "somewhat less likely based on history[] [and] scans." Id. In the afternoon, Petitioner received a second dose of Solu-Medrol and reported "feeling increased strength overall." Id. at 334, 345. Bloodwork taken that afternoon was negative for neuromyelitis optica ("NMO") Immunoglobulin G ("IgG") antibodies.[14] Id. at 394. Dr. Woody saw Petitioner that evening and documented Petitioner "continue[d] to feel better and stronger" with "less fatigue." Id. at 342.

Petitioner received her third treatment of Solu-Medrol on the morning of June 13, 2019. Pet. Ex. 4 at 322. Hospitalist Stephen Matthew Furlow, M.D.,[15] summarized Petitioner's hospital course: Petitioner "present[ed] to the hospital with worsening bilateral leg strength and difficulty walking after [a] recent HPV vaccination. . . . Neurology evaluated her and felt [TM] was very likely and initiated high-dose steroid treatment which rapidly resolved her weakness." Id. Following completion of her recommended steroid dosing, she was "walking well independently" and reported she "fe[lt] her strength [was] pretty much back to normal." Id. Petitioner was cleared for discharge with a diagnosis of "[p]resumed [a]cute [TM]." Id.

Petitioner returned to the ED on June 17 for a persistent headache. Pet. Ex. 4 at 268. She received a blood patch to seal a CSF leak following her lumbar puncture. Id. at 270, 272.

On June 20, 2019, Petitioner saw her PCP following her hospitalization. Pet. Ex. 2 at 8-11. Petitioner reported her clinical course and subsequent diagnosis of TM and a CSF leak. Id. at 11. On examination, Petitioner had mild orthostasis (low blood pressure) but was asymptomatic on standing, as well as mild dehydration. Id. The HPV vaccination was listed under allergies. Id. at 9.

On July 5, 2019, Petitioner presented to neurologist James M. Winkley, M.D. Pet. Ex. 5 at 189. Petitioner reported her lower extremity weakness had improved by 85%. Id. She "[felt] overall drained," her trunk felt heavy when sitting, and she had occasional numbness and tingling in her left calf. Id. She "had band like sensation [at T10] with increased sensitivity." Id. Dr. Winkley reviewed Petitioner's hospitalization records, noting work up was unremarkable, and conducted an examination that was normal. Id. at 189, 191-97. Assessment was "[p]resumed

[14] NMO IgG autoantibodies are associated with TM and other CNS and autoimmune disorders associated with TM. Resp. Ex. B at 3; infra note 18.

[15] On June 26, 2019, Dr. Furlow responded to an internal email from hospital administration requesting clarification on Petitioner's condition. Pet. Ex. 4 at 531. Dr. Furlow responded, specifying Petitioner's condition as "[a]cute [TM] possibly due to recent [HPV] vaccination." Id.

[a]cute [TM]" with "[symptoms] of presumed T10 [TM]." Id. at 197. Dr. Winkley recommended "[w]atchful waiting." Id.

On July 19, 2019, Petitioner returned to her PCP complaining of continued "severe generalized fatigue and headache," as well as neck pain. Pet. Ex. 2 at 3. On examination, Petitioner had tight neck muscles, for which she was prescribed Flexeril. Id. at 6-7. Bloodwork was ordered and showed Petitioner's Epstein-Barr virus ("EBV")[16] antibody titers were elevated, including nuclear antigen (358.0; reference range: 0-21.9) and early antigen D (35.2; reference range: 0-10.9). Id. at 7, 26-35.

On October 31, 2019, Petitioner presented to the ED complaining of "lower back pain" that "has been constant for the past few days. She describe[d] her pain as a shooting sensation that radiate[d] into her bilateral legs." Pet. Ex. 4 at 218. Petitioner also reported bilateral leg numbness. Id. She indicated her current pain was located at the site of her lumbar puncture. Id. On examination, Petitioner had lumbar paravertebral tenderness, "[s]cant area of possible swelling vs adipose tissue," no cellulitis, good push/pull movement of her feet, and a painful range of motion of her left leg that she attributed to her chronic knee condition. Id. at 220. A repeat MRI of Petitioner's lumbar spine, with and without contrast, was unremarkable. Id. at 223, 233-34. Bloodwork showed a slightly elevated C-reactive protein ("CRP")[17] (0.99; reference range: 0.00-0.50). Id. at 222, 229. Petitioner was advised to return to her PCP for an autoimmune work-up. Id. at 221.

Petitioner returned to neurologist Dr. Winkley on December 2, 2019, complaining of low back pain and stiffness, "continued severe fatigue," and intermittent numbness and tingling in her left lower leg. Pet. Ex. 5 at 165. Petitioner noted her October ED visit and reported her symptoms improved on prednisone. Id. Dr. Winkley documented hyperreflexia (3+ reflexes in bilateral patellar and Achilles) on examination. Id. at 168-69. He ordered a repeat MRI series that was unremarkable. Id. at 146, 152, 171-72. He also screened for antibodies to NMO Aquaporin-4 ("AQP4") and myelin oligodendrocyte glycoprotein ("MOG"),[18] which were negative. Id. at 171; Pet. Ex. 4 at 95-97.

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[16] EBV is "a virus of the genus Lymphocryptovirus that causes infectious mononucleosis." Human Herpesvirus 4, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=80849 (last visited Mar. 18, 2025).

[17] CRP is "a globulin that forms a precipitate with the somatic C-polysaccharide of the pneumococcus in vitro; it is the most predominant of the acute-phase proteins." C-Reactive Protein, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=100489 (last visited Mar. 18, 2025).

[18] Testing for NMO AQP4 and MOG is done in "patients with suspected TM of unknown etiology," as these antibodies are associated with CNS disorders that can cause TM and systemic autoimmune disorders with TM as the initial presenting event. Pet. Ex. 34 at 3-4, 10-11; see also Pet. Ex. 11 at 4 (noting MOG and AQP4 are associated with TM); Resp. Ex. B at 11 ("Autoantibodies, such as anti-MOG or [AQP4], are typically found when [TM] is associated with an autoimmune process.").

In February 2020, Petitioner returned to the ED and also saw Dr. Winkley for continued complaints of headaches, low back pain, and numbness and tingling in her left upper and lower extremities. Pet. Ex. 4 at 110-13; Pet. Ex. 5 at 100-06, 118, 127. Petitioner was treated with increasing doses of Trileptal[19] and a tapering dose of oral steroids. Pet. Ex. 5 at 59, 99, 105. Petitioner was positive for antinuclear antibodies ("ANA")[20] and electromyography ("EMG")/ nerve conduction study ("NCS") of her left leg was normal. Id. at 70-73; Pet. Ex. 4 at 70.

Petitioner had a telemedicine visit with Dr. Winkley on April 17, 2020. Pet. Ex. 5 at 27. Petitioner no longer had numbness in her left leg. Id. She was taking the maximum dosage of Trileptal and her symptoms were 85% improved. Id. She had mild back pain when walking and her headaches were improving with treatment. Id. at 27, 30. Dr. Winkley's assessment was periodic headache syndrome, not intractable. Id. at 30. Petitioner was directed to follow up in six months, but appears to have not done so. Id.; see also Pet. Ex. 8 at 87 (noting no recent neurology follow-up at a visit in February 2021).

On February 3, 2021, Petitioner established care with a new PCP. Pet. Ex. 8 at 87. Petitioner complained of occasional low back pain and left leg weakness and numbness since her TM diagnosis in 2019. Id. Petitioner reported Trileptal and steroids helped with leg weakness and numbness. Id. She was no longer taking Trileptal. Id. Petitioner also reported "bilateral wrist pain, left elbow pain, upper shoulder and neck pain, and left knee pain for years." Id. Petitioner also noted she was "always tired," had "pain [] to the touch," and "fe[lt] achy all over" with easy bruising. Id. Examination revealed diffuse tenderness to palpation in elbows, wrists, knees, ankles, back, shoulders, and neck. Id. at 88. Petitioner was diagnosed with chronic fatigue, arthralgia, and myalgia. Id. at 99. A repeat ANA was negative, and Petitioner's CRP was "slightly elevated." Id. at 74. Petitioner returned on March 16, 2021 for "all over pain." Id. at 54. She was prescribed a steroid taper to help with symptoms. Id. at 56.

On April 6, 2021, Petitioner presented to rheumatologist Jeffrey Neal, M.D., and complained of diffuse pain and tenderness and fatigue "for at least 10 years." Pet. Ex. 7 at 5. Dr. Neal noted that multiple workups with a neurologist revealed "no persistent neurologic effect of the [TM]." Id. On examination, Dr. Neal noted mild hyperreflexia and "18/18 classic fibromyalgia tender points are very tender." Id. at 7-8. He diagnosed Petitioner with fibromyalgia and ordered a complete rheumatological work-up, which was negative. Id. at 8-9, 14. He noted that Petitioner had an "[a]pparent complete solution" of her TM, and "[o]ther than mild hyperreflexia, she ha[d] no notable neurologic abnormalities." Id. at 9.

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[19] Trileptal (oxcarbazepine) is "an anticonvulsant used in the treatment of partial seizures." Oxcarbazepine, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=36142 (last visited Mar. 18, 2025).

[20] ANA are "antibodies directed against nuclear antigens; ones against a variety of different antigens are almost invariably found in systemic lupus erythematosus and are frequently found in rheumatoid arthritis, scleroderma (systemic sclerosis), Sjögren syndrome, and mixed connective tissue disease." Antinuclear Antibodies, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=56804 (last visited Mar. 18, 2025).

On August 16, 2021, Kharri Hollingshead, Physician Assistant ("PA"), wrote a letter stating Petitioner "has a history of [TM] secondary to HPV vaccination. This is a rare and significant complication where the patient develops inflammation of the spinal cord. Due to this history, it is strongly recommended that [Petitioner] does not obtain the COVID-19 vaccination at this time." Pet. Ex. 29 at 294.

No additional relevant records were filed.

C. Expert Reports[21]

1. Petitioner's Expert, Dr. Lawrence Steinman[22]

a. Background and Qualifications

Dr. Steinman is board certified in neurology and has practiced neurology at Stanford University for over 40 years. Pet. Ex. 11 at 1; Pet. Ex. 12 at 1-2. He received his B.A. from Dartmouth College in 1968 and his M.D. from Harvard University in 1973. Pet. Ex. 12 at 1. Thereafter, he completed a surgery internship, pediatrics residency, and pediatric and adult neurology residency at Stanford University Hospital, as well as three fellowships, including one in clinical immunology. Id. Dr. Steinman is currently a Professor at Stanford University. Id. Dr. Steinman "is actively involved in patient care" and "ha[s] cared for hundreds of adults and children with various forms of neuroinflammatory diseases including [acute demyelinating encephalomyelitis ("ADEM")], optic neuritis, [GBS], chronic inflammatory neuropathy (CIDP), [TM], inflammatory neuropathy, [NMO,] and [MS]." Pet. Ex. 11 at 1. He has authored or co-authored over 600 publications. Pet. Ex. 12 at 5-49. Dr. Steinman has authored papers on molecular mimicry, as demonstrated by his CV. See id. One of Dr. Steinman's specialties is in the area of MS, and he has received a Charcot Prize for Lifetime Achievement due to his research in MS. Pet. Ex. 112 at 3. In 2015, he was elected to the National Academy of Sciences. Id.

[21] Although the undersigned has reviewed all expert reports, for the sake of brevity, this Decision does not include every detail of the experts' opinions. Instead, the undersigned focuses on the experts' material opinions, as they relate to the relevant issues.

[22] Dr. Steinman provided two expert reports. Pet. Exs. 11, 32.

b. Opinion

Dr. Steinman opined "more likely than not Petitioner had a recall response[23] to the [HPV] vaccine[] and developed [TM]." Pet. Ex. 11 at 1.

i. Diagnosis

Dr. Steinman opined, by a preponderance of evidence, that the more likely than not diagnosis for Petitioner was TM. Pet. Ex. 11 at 5. He acknowledged that "definitive evidence for [TM] [was] absent." Id.; see also Pet. Ex. 34 at 8 (noting "some patients presenting with TM may not fulfill all of the [diagnostic] criteria" and "the absence of inflammatory markers does not rule out TM"). However, he maintained Petitioner's working diagnosis was always suspected or presumptive TM. Pet. Ex. 11 at 5.

He asserted that her early treatment with corticosteroids prior to definitive diagnosis "may have masked evidence that might have appeared had the disease worsened." Pet. Ex. 11 at 5. Dr. Steinman noted that he would have also chosen to treat Petitioner with corticosteroids prior to diagnostic testing results. Id.; Pet. Ex. 32 at 1-2; see also Pet. Ex. 34 at 15-16.

He disagreed with Dr. Jamieson's opinion that Petitioner's rapid recovery did not support a diagnosis of TM. Pet. Ex. 32 at 2. Dr. Steinman has seen rapid resolution of inflammatory lesions on MRI after one dose of Solu-Medrol. Id. For support, he cited a case report from Kennedy and Weir[24] that discussed a TM patient treated with steroids who experienced a rapid clinical and electrophysiological recovery within 24 hours. Id. (citing Pet. Ex. 33). Dr. Steinman also cited a 2023 UpToDate article from Greenberg that "suggest[ed] high-dose intravenous glucocorticoid treatment for patients with acute idiopathic TM . . . should be initiated as soon as possible; there are relatively few contraindications. Thus, a clinician does not need to wait for the workup to be complete before initiating therapy." Id. (quoting Pet. Ex. 34 at 15); see also Resp. Ex. A-3 at 3 ("[F]or clinical management . . . of patients with suspected [acute TM], it may not be prudent to wait until the nadir is reached. Rather, treatment may be initiated with continued observation to determine if the patient ultimately meets all the criteria.").

Dr. Steinman disagreed with Dr. Jamieson's FND diagnosis, noting such diagnosis was not mentioned or discussed by Petitioner's treating physicians. Pet. Ex. 32 at 1, 3-4. He also

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[23] A recall response refers to a secondary immune response, which is "the immune response occurring on the second and subsequent exposures to an antigen; compared to a primary immune response, the lag period is shorter, the peak antibody titer is higher and lasts longer, IgG production predominates, the antibodies produced have a higher affinity for the antigen, and a much smaller dose of the antigen is required to initiate the response." Immune Response, Secondary, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=103686 (last visited Mar. 18, 2025).

[24] P.G.E. Kennedy & A.I. Weir, Rapid Recovery of Acute Transverse Myelitis Treated with Steroids, 64 Postgraduate Med. J. 384 (1988).

disagreed that Petitioner's TM symptoms could be explained by fibromyalgia since it was not considered a diagnosis until Petitioner saw Dr. Neal in 2021, years after vaccination. Id. at 3-4.

ii. Althen Prong One

Dr. Steinman opined the HPV vaccine can cause TM via molecular mimicry due to homology between the HPV vaccine (specifically, L1 protein in HPV Type 11) and myelin basic protein ("MBP"). Pet. Ex. 11 at 5, 7. He focused on MBP "because it is the most abundant myelin protein and because Abramsky and Teitelbaum[25] showed that [MBP], then called myelin basic encephalitogenic protein[,] was a target of the immune attack in acute [TM]." Id. at 5 (citing Pet. Ex. 13).

TM has been reported to occur after vaccination and infection, with molecular mimicry as the postulated mechanism of injury. Pet. Ex. 34 at 2, 5. To explain molecular mimicry, Dr. Steinman cited an illustration in a 1993 article he authored.[26] Pet. Ex. 11 at 6 (citing Pet. Ex. 14 at 4). In that paper, he explained "T cells recognize foreign antigens when they are presented by the [human leukocyte antigen ("HLA")[27]] molecules of the immune system," and such "foreign antigen may resemble antigen[s] produced by the body," which "provokes the T cells to attack body tissues that contain the self-antigens." Pet. Ex. 14 at 4. He also quoted a study from Birnbaum et al.[28] that found "autoimmune T cells have the ability to be activated by immunogens encountered in the environment, which may serve as the trigger for the initiation of autoimmunity." Pet. Ex. 11 at 7 (quoting Pet. Ex. 17 at 13).

Next, Dr. Steinman cited to the HPV package insert to show the ingredients contained in the vaccine at issue. Pet. Ex. 11 at 7 (citing Pet. Ex. 18 at 12-13 (package insert)). The HPV vaccine "is a non-infectious recombinant 9-valent vaccine prepared from the purified virus-like particles (VLPs) of the major capsid (L1) protein of HPV Types 6, 11, 16, 18, 31, 33, 45, 52, and 58. The L1 proteins are produced by separate fermentations using recombinant *Saccharomyces cerevisiae* and self-assembled into VLPs." Pet. Ex. 18 at 12.

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[25] Oded Abramsky & Dvora Teitelbaum, The Autoimmune Features of Acute Transverse Myelopathy, 2 Annals Neurology 36 (1977). Dr. Steinman does not detail or discuss this study. And, as Dr. Matloubian noted, this article is from 1977 and no other literature provided from Dr. Steinman is supportive. See Resp. Ex. B at 14.

[26] Lawrence Steinman, Autoimmune Disease, 269 Sci. Am. 106 (1993).

[27] HLA are "histocompatibility antigens governed by genes of the HLA complex (the human major histocompatibility complex), a region on the short arm of chromosome 6 containing several genetic loci, each having multiple alleles." Human Leukocyte Antigens, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=56923 (last visited Mar. 18, 2025).

[28] Michael E. Birnbaum et al., Deconstructing the Peptide-MHC Specificity of T Cell Recognition, 157 Cell 1073 (2014). For the supplemental data referenced in this article, see Resp. Ex. B-19.

Dr. Steinman relied on a study from Wucherpfennig and Strominger[29] to explain how the HPV vaccination could trigger inflammation in the CNS similar to TM via molecular mimicry. Pet. Ex. 11 at 7-9 (citing Pet. Ex. 19). According to Dr. Steinman, Wucherpfennig and Strominger "looked for sequences in viruses and bacteria that had amino acid motifs that included the main binding components of [MBP] to the major histocompatibility complex (MHC)"[30] and found "some human clones respond to [HPV]." Id. (citing Pet. Ex. 19). "Their analysis allowed for certain substitutions at different positions, to see how much variability was possible in a mimic, that would still allow recognition by a [MBP] reactive T cell clone." Id. at 8. This study, however, used the live HPV virus, not virus-like particles from the HPV vaccine, which does not contain a live viral component. See Pet. Ex. 19. Furthermore, the sequence similarity reported by the authors was with L2 protein of HPV Type 7 virus, which is not contained in the vaccine at issue here. Id. at 6 tbl.2. Dr. Steinman did not explain how this sequence similarity correlates to the sequence homology he proposes with the L1 protein of HPV Type 11 in the vaccine.

Next, he opined the sequence for MBP[31] and L1 protein for HPV Type 11[32] contained five identical amino acids, three of which were consecutive "at critical binding points."[33] Pet. Ex. 11 at 9-10.

According to Dr. Steinman, substitutions at two positions are "tolerated—that is these substitutions are recognized by a [MBP] reactive T cell close, albeit weakly"—which would lead

[29] Kai W. Wucherpfennig & Jack L. Strominger, Molecular Mimicry in T Cell-Mediated Autoimmunity: Viral Peptides Activated Human T Cell Clones Specific for Myelin Basic Protein, 80 Cell 695 (1995).

[30] MHC are "the genes determining the major histocompatibility antigens, in all species a group of closely linked multiallelic genes located in a small region on one chromosome." Major Histocompatibility Complex, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=66341 (last visited Mar. 18, 2025).

[31] This sequence is "ENPVVHFFKNIVTPRTP." Pet. Ex. 11 at 9.

[32] This sequence is "QMFARHFFNRAGTVGEP." Pet. Ex. 11 at 9.

[33] Dr. Steinman cited Birnbaum et al. to support this assertion but the undersigned does not find it informative. See Pet. Ex. 17.

to "[seven] out of 12 consecutive amino acids with either exact or acceptable substitutions[34] that [would] allow stimulation of a human MBP reactive T cell clone." Pet. Ex. 11 at 10. Dr. Steinman noted "[t]hese acceptable amino acids occur at positions that include many major HLA and [T-cell receptor ("TCR")] contact sites for T cell recognition of MBP in humans." Id.

Dr. Steinman opined this sequence homology is significant and has been shown to trigger experimental autoimmune encephalomyelitis ("EAE") in an experimental model of ADEM/TM. Pet. Ex. 11 at 10. For support, he cited the Gautam et al.[35] studies for the proposition that autoimmune encephalomyelitis could be induced with only five amino acids identical to MBP.[36] Id. at 10-13 (citing Pet. Exs. 21-23). He also cited other studies related to MS that showed amino acid substitutions are "allowable," which can then lead to greater potential for homology. Id. at 15-20 (citing Pet. Exs. 20, 24-25).

Dr. Steinman concluded that his theory "is based on molecular mimicry between the components of the [HPV] vaccine and proteins known to be shared with neuronal antigens attacked by the human immune system in polyneuropathy." Pet. Ex. 11 at 23; Pet. Ex. 32 at 6. However, polyneuropathy is a neuropathy of peripheral nerves in the peripheral nervous system ("PNS"),[37] while here, TM is the alleged injury and it involves the spinal cord within the CNS.

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[34] For these substitutions, and the studies on which Dr. Steinman relied upon to assert substitutions are "allowable," see Pet. Ex. 11 at 10, 15-20; Pet. Ex. 20 (Stefan Hausmann et al., Structural Features of Autoreactive TCR That Determine the Degree of Degeneracy in Peptide Recognition, 162 J. Immunology 338 (1999)); Pet. Ex. 24 (Kai W. Wucherpfennig et al., Recognition of the Immunodominant Myelin Basic Protein Peptide by Autoantibodies and HLA-DR2-Restricted T Cell Clones from Multiple Sclerosis Patients: Identity of Key Contact Residues in the B-Cell and T-Cell Epitopes, 100 J. Clinical Investigation 1114 (1997)); Pet. Ex. 25 (Kai W. Wucherpfennig et al., Structure of Human T-Cell Receptors Specific for an Immunodominant Myelin Basic Protein Peptide: Positioning of T-Cell Receptors on HLA-DR2/Peptide Complexes, 92 Proc. Nat'l Acad. Scis. 8896 (1995)).

[35] Anand M. Gautam et al., A Viral Peptide with Limited Homology to a Self Peptide Can Induce Clinical Signs of Experimental Autoimmune Encephalomyelitis, 161 J. Immunology 60 (1998); Anand M. Gautam et al., Minimum Structural Requirements for Peptide Presentation by Major Histocompatibility Complex Class II Molecules: Implications in Induction of Autoimmunity, 91 Immunology 767 (1994); Anand M. Gautam et al., A Polyalanine Peptide with Only Five Native Myelin Basic Protein Residues Induces Autoimmune Encephalomyelitis, 176 J. Experimental Med. 605 (1992). Dr. Steinman is a named author in all of these papers.

[36] In Dr. Steinman's report, he noted three recent publications showed "this degree of homology may be at the foundations of how neuroinflammation can be triggered by a piece of a virus and how that can trigger MS." Pet. Ex. 11 at 13. He then went on to briefly discuss these publications; however, none were filed. See id. at 13-15.

[37] Polyneuropathy, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=40203 (last visited Mar. 18, 2025).

iii. Althen Prong Two

Dr. Steinman opined, "by a preponderance of evidence," Petitioner's "[HPV] vaccin[ation] on June 7, 2019 was the more likely than not cause of Petitioner's [TM]." Pet. Ex. 11 at 23.

Dr. Steinman summarized Petitioner's clinical course. Pet. Ex. 11 at 20-21. Petitioner received her third HPV vaccination on June 7, 2019. Id. at 20. On June 10, she presented to the ED with complaints of extremity weakness and fatigue. Id. Petitioner reported itching and fatigue shortly after vaccination, which similarly occurred with her two prior HPV vaccinations. Id. However, these symptoms did not resolve and began to worsen. Id. at 20-21. Two days prior to the ED (June 8), her symptoms worsened and the day prior to the ED (June 9), she began experiencing extremity weakness, with falls on both June 9 and June 10. Id. at 21.

He opined "[e]xtensive studies for infectious, neoplastic, and structural causes for TM failed to provide a satisfactory alternative cause." Pet. Ex. 11 at 5.

iv. Althen Prong Three

Next, Dr. Steinman opined that Petitioner's onset of TM was within one to two days after vaccination. Pet. Ex. 11 at 20. To support such timing fulfills prong three, Dr. Steinman cited Schonberger et al.[38] to show cases of GBS post-influenza vaccine were included in the interval of day 0 (day of vaccination) to day one (day after vaccination). Id. at 21, 24 (citing Pet. Ex. 26). However, Schonberger et al. did not discuss TM, a CNS disease, and instead focused on GBS, a PNS disease. And Schonberger et al. did not discuss HPV vaccinations.

[38] Lawrence B. Schonberger et al., Guillain Barré Syndrome Following Vaccination in the National Influenza Immunization Program, United States, 1976-1977, 110 Am J. Epidemiology 105 (1979). Petitioner did not file this article, and instead filed an article from Haber et al. that discussed Schonberger et al. and conducted its own study of Vaccine Adverse Event Reporting System ("VAERS") reports of GBS post-influenza vaccination from July 1990 through June 2003. See Pet. Ex. 26 (Penina Haber et al., Guillain-Barré Syndrome Following Influenza Vaccination, 292 JAMA 2478 (2004)). Neither article discusses TM or the HPV vaccine.

Dr. Steinman asserted a recall response occurred here and was responsible for the rapid onset.[39] Pet. Ex. 11 at 22. He cited to Lai et al.,[40] asserting a recall response can occur as early as six hours after antigenic challenge. Id. at 22-23 (citing Pet. Ex. 35). Dr. Steinman does not discuss this study or further explain how it applies to the facts and circumstances of this case.[41]

He also cited to the 2012 Institute of Medicine ("IOM") report,[42] which discussed the latency between antigen exposure and development of an adaptive immune response. Pet. Ex. 11 at 22 (citing Pet. Ex. 28 at 86-87). As the IOM explained, "[f]or both B and T cells in a typical immune response to an antigen exposure, the latency between the first (primary) exposure and development of the primary response is characterized by a lag phase, logarithmic phase, and plateau phase." Pet. Ex. 28 at 86-87. "The lag phase is characterized by the initial activation of B and T cells upon encounter with the antigen for which they are specific, and this triggers the cells' differentiation into effector and memory cells." Id. at 87. With a primary exposure, the lag phase between exposure of an antigen and the logarithmic phase, which "is characterized by an increase in serum antibody levels that classically is logarithmic," is thought to be between four to seven days, depending on route of exposure and the antigen. Id. The third phase, the plateau phase, "is characterized by the maintenance of peak antibody levels for a length of time that is followed by a decline in the serum antibody levels." Id. Overall, for a first/primary exposure, the latency period "between [] exposure and development of the primary antibody response is [seven] to 10 days." Id.

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[39] The undersigned does not discuss Dr. Steinman's references to tuberculin tests, as these discussions are not relevant since they do not address the vaccine, injury, or theory posited here. Dr. Steinman's discussion of tuberculin tests was also rejected as not relevant in at least one other decision. See Greenslade v. Sec'y of Health & Hum. Servs., 14-1140V, 2024 WL 3527665, at *39 (Fed. Cl. Spec. Mstr. June 28, 2024). Nor does the undersigned discuss Dr. Steinman's references to past Vaccine Program cases, as they are not material to this case because they concern a different vaccine and/or injury and they are not binding on the undersigned. See Hanlon v. Sec'y of Health & Hum. Servs., 40 Fed. Cl. 625, 630 (1998), aff'd, 191 F.3d 1344 (Fed. Cir. 1999); Boatmon v. Sec'y of Health & Hum. Servs., 941 F.3d 1351, 1358 (Fed. Cir. 2019).

[40] Wendy Lai et al., Transcriptional Control of Rapid Recall by Memory CD4 T Cells, 187 J. Immunology 133 (2011).

[41] Dr. Steinman stated he cited Lai et al. because he cited it in another Vaccine Program case with a short onset period and that special master found it persuasive. Pet. Ex. 11 at 22-23; Pet. Ex. 32 at 5. However, that case concerned an injury of small fiber neuropathy, not TM. Dr. Steinman does not explain how a small fiber neuropathy case is applicable here. Therefore, the undersigned does not find this case and citation to be pertinent to this present case. And again, it is not binding on the undersigned. See Hanlon, 40 Fed. Cl. at 630 (1998); Boatmon, 941 F.3d at 1358.

[42] Inst. of Med., Adverse Effects of Vaccines: Evidence and Causality (Kathleen Stratton et al. eds., 2012). Portions of this text were also filed as Resp. Ex. B-8.

Here, however, a recall response is at play due to Petitioner's previous HPV vaccinations. Pet. Ex. 11 at 22; Pet. Ex. 28 at 87. In a secondary/recall response, memory B and T cells previously developed during primary exposure and response. Pet. Ex. 28 at 87. Because of this, the latency between subsequent/secondary exposures and development of an antibody response is shorter. Id. "The lag phase is generally [one] to [three] days[,] [and] the logarithmic phase of the secondary antibody response occurs over the next [three] to [five] days." Id. This results in a latency period (including both the lag and logarithmic phases) ranging from four to eight days. See id. Dr. Steinman did not explain how such a latency period conforms to the one to two day onset here.

2. Respondent's Expert, Dr. Dara G. Jamieson[43]

a. Background and Qualifications

Dr. Jamieson is a board-certified neurologist. Resp. Ex. A at 1; Resp. Ex. A-1 at 2. She received her medical degree from the University of Pennsylvania, followed by a neurology residency and a cerebrovascular fellowship at the University of Pennsylvania Hospital. Resp. Ex. A-1 at 1. Dr. Jamieson was a practicing neurologist for 32 years before transiting to a teaching appointment. Resp. Ex. A at 1. She is currently a Clinical Associate Professor of Neurology at Weill Cornell Medicine, where she teaches medical students, residents, and fellows. Id. Dr. Jamieson has lectured extensively on multiple neurological topics. Id. She serves as an editor and reviewer for several neurology journals. Id. at 1-2. Dr. Jaimeson has authored or co-authored numerous neurology papers, chapters, review articles, and books. Id. at 2; Resp. Ex. A-1 at 10-14.

b. Diagnosis Opinion

Overall, Dr. Jamieson opined Petitioner "did not develop [TM], and she did not have residual effects or complications of the leg weakness, which was not due to [TM], for more than six months."[44] Resp. Ex. A at 9. Instead, Dr. Jamieson opined Petitioner developed a FND following her HPV vaccination. Id. at 10. Dr. Jamieson concluded "[t]here is no immunological causative association between the HPV vaccine and [Petitioner's] transient leg weakness due to FND." Id. at 14.

[43] Dr. Jamieson submitted two expert reports. Resp. Exs. A, C. She addressed diagnosis and did not opine as to causation.

[44] Although Respondent raised a six-month severity issue in his Rule 4(c) report, Respondent failed to raise this issue in briefing. See Resp. Rept. at 11-12; Joint Submission; Resp. Response. The undersigned will not address this issue as it appears abandoned by Respondent. Even if it had been raised, the undersigned would have rejected it since the medical records show a clinical course exceeding six months. See, e.g., Pet. Ex. 5 at 27 (noting, in April 2020, Petitioner was taking the maximum dosage of Trileptal and her symptoms were 85% improved). Thus, in April 2020, Petitioner had not fully recovered.

Dr. Jamieson detailed Petitioner pre-vaccination and post-vaccination medical history. Resp. Ex. A at 2-10; Resp. Ex. C at 1-3. She explained Petitioner had a "long standing psychiatric disease [history] [and] developed subjective symptoms of fatigue and leg weakness starting on the day that she was given her third vaccine against HPV," June 7, 2019. Resp. Ex. A at 9. Three days later, on June 10, she presented to the ED "with variable severity of subjective leg weakness, without bladder or bowel complaints." Id. Petitioner's "bilateral leg weakness appeared to be profound on [June 10] . . . , but by the next day it only appeared to be mild, and by [June 12], her complaints of weakness had resolved." Id.

Following her hospitalization, and subsequent post-dural puncture headache that required a blood patch, Dr. Jamieson asserted Petitioner had no further symptoms related to her complaints immediately after her third HPV vaccination. Resp. Ex. A at 9-10. Petitioner continued to complain of her pre-existing issues (e.g., headaches); however, Dr. Jamieson opined these complaints were "anatomically diffuse and inconsistent with a spinal cord lesion." Id. at 10. Then, in 2021, Petitioner was diagnosed with fibromyalgia. Id.; Resp. Ex. C at 3.

i. Transverse Myelitis

Dr. Jamieson discussed TM and explained how to diagnose TM. Resp. Ex. A at 10-11. TM is a CNS condition with "spinal cord dysfunction resulting in weakness, sensory loss below the level of the lesion [in the spinal cord], and impairment in autonomic functioning including bladder and bowel dysfunction." Id. at 10; see also Resp. Ex. A-3 at 1 (defining TM as "a focal inflammatory disorder of the spinal cord, resulting in motor, sensory, and autonomic dysfunction"). Evaluation of a patient presenting with symptoms consistent with a thoracic spinal cord lesion includes neurological examination to document lower extremity weakness and sensory loss; MRIs to localize spinal cord lesions and show the extent of inflammation; bloodwork to confirm inflammation (white blood cells); and a lumbar puncture to show elevated protein in the CSF. Resp. Ex. A at 10; see Resp. Ex. A-3 at 1 ("There is often a clearly defined [level] of sensory dysfunction, and spinal MRI and lumbar puncture often show evidence of acute inflammation."). In TM, an MRI of the cervical or thoracic spine "shows a spinal cord lesion extending for more than two spinal column segments and involving more than two thirds of the transverse extent of the spinal cord." Resp. Ex. A at 10.

TM may manifest as rapid-onset, severe paraparesis or quadriparesis with areflexia or hyperreflexia. Resp. Ex. A-6 at 2.[45] Presence of hyperreflexia confirms a CNS cause, rather than a PNS cause, of muscle weakness. Id. At maximal deficit, "approximately 50% of patients have lost all movements of their legs, virtually all patients have bladder dysfunction, and 80 to 94% of patients have numbness, paresthesias, or band-like dysesthesias." Resp. Ex. A-3 at 1. "Rapid progression of symptoms, back pain, and spinal shock predict poor recovery." Id.

Dr. Jamieson cited the diagnostic inclusion criteria for TM from the TM Consortium Working Group to establish that "a specific combination of symptoms, findings on neurological

[45] Elliot M. Frohman & Dean M. Wingerchuck, Transverse Myelitis, 363 New Eng. J. Med. 564 (2010).

examination, and abnormalities on testing" are required for TM diagnosis. Resp. Ex. A at 11 (citing Resp. Ex. A-3 at 2 tbl.1). The diagnostic inclusion criteria[46] for TM are,

1. Development of sensory, motor, or autonomic dysfunction attributable to the spinal cord;
2. Bilateral signs and/or symptoms;
3. Clearly defined sensory level;
4. Exclusion of extra-axial compressive etiology by neuroimaging;
5. Inflammation within the spinal cord demonstrated by CSF pleocytosis[47] or elevated IgG index or gadolinium enhancement; and
6. Progression to nadir between [four] hours and 21 days following the onset of symptoms.

Id. (citing Resp. Ex. A-3 at 2 tbl.1).

Based on this criteria, Dr. Jamieson opined Petitioner's clinical presentation, diagnostic imaging, and laboratory testing was not consistent with TM. Resp. Ex. A at 11. First, as to clinical presentation, Dr. Jamieson noted Petitioner's symptoms of leg weakness were "very brief" and rapidly resolved within two to three days, which is uncharacteristic of TM or a demyelinating illness. Id.; see also Resp. Ex. A-6 at 2 ("Most of the recovery occurs over the course of the first [three] months after the event, although improvement may continue for a year or longer.").

Next, Dr. Jamieson opined Petitioner failed to meet clinical diagnostic criteria as Petitioner did not have autonomic dysfunction or a clearly defined sensory level. Resp. Ex. A at 11. "Autonomic symptoms consist variably of increased urinary urgency, bowel or bladder incontinence, difficulty or inability to void, incomplete evacuation, or bowel constipation." Resp. Ex. A-3 at 1.

Petitioner also lacked objective diagnostic markers of TM. Resp. Ex. A at 11. There was no evidence of CSF inflammation (no pleocytosis, elevated protein, or oligoclonal bands) and no lesions seen on MRI. Id. Dr. Jamieson maintained that "[n]either [Petitioner's] one dose of steroids, which is routinely given when [TM] is even suspected, nor the timing of the testing relative to the onset of her symptoms, caused the absence of evidence of [TM] on imaging or in her CSF." Id. She agreed steroid treatment should be initiated as soon as possible, prior to workup being completed. Resp. Ex. C at 4 (citing Pet. Ex. 34 at 15). However, she opined a short course treatment of steroids without more is not "probative" evidence of a TM diagnosis. Id. "[O]nce the workup [was] complete, without evidence to support the diagnosis of [TM], the fact that steroids were given initially does not endorse a discarded diagnosis for [Petitioner]." Id.

[46] "[S]ome patients presenting with TM may not fulfill all of the above criteria. . . . [T]he absence of inflammatory markers does not rule out TM." Pet. Ex. 34 at 8.

[47] Pleocytosis refers to the "presence of a greater than normal number of cells in the [CSF]." Pleocytosis, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=39556 (last visited Mar. 18, 2025).

Dr. Jamieson contended retaining a preliminary diagnosis of TM after it was contracted was improper.[48] Id.

Dr. Jamieson concluded that although TM was suspected, and Petitioner was treated with steroids consistent with treatment for TM, all diagnostic testing for TM was negative. Resp. Ex. A at 9-11. Even though testing was conducted after "only one dose of steroids[,] . . . [Petitioner] was still exhibiting leg weakness," and therefore, there would have been evidence of TM on testing. Id. Thus, she opined Petitioner did not have TM. Id.

ii. Functional Neurologic Disorder

Instead of TM, Dr. Jamieson asserted that Petitioner had a transient episode of FND following her third HPV vaccination. Resp. Ex. A at 12, 14.

Dr. Jamieson explained an FND "denotes an impairment of the function of a nervous system that is actually capable of normal functioning. . . . The disorder describes the presence of atypical neurologic-type symptoms that do not conform to the known anatomic and physiologic constructs that support a recognized neurologic diagnosis or neuroanatomic localization." Resp. Ex. A at 12. A psychological stressor can convert into physical symptoms, although previous traumatic and/or stressful events are not prerequisites, only risk factors, for the development of FND. Id.; see Resp. Ex. A-7 at 1.[49] Risk factors for FND include "adverse experiences during childhood; a previous physical injury; panic attacks; mood disorders such as anxiety or depression; and dissociation, which is a feeling of disconnection from either the surrounding world (derealization) or one's own body (depersonalization)." Resp. Ex. A at 12; see also Resp. Ex. A-8 at 6;[50] Resp. Ex. A-9 at 4-5.[51] A physical injury or a stressful life event may trigger an acute onset of FND. Resp. Ex. A at 12. Dr. Jamieson maintained that "FNDs can have clinical features that are bizarre and diagnostic testing that is unrevealing, [and] FND is not just a diagnosis of exclusion" and "should be diagnosed based on positive features of internal inconsistency or incongruity in the clinical course of symptoms or on the neurological examination." Id.; see also Resp. Ex. A-9 at 8.

To support a diagnosis of FND for Petitioner, Dr. Jamieson noted Petitioner had multiple predisposing factors for FND (including mood disorders, panic attacks, childhood trauma causing PTSD, and derealization). Resp. Ex. A at 13. After the vaccination, her mother

[48] Dr. Jamieson asserted "Dr. Steinman's emphasis on the diagnosis of suspected [TM] in the medical records is an example of anchoring bias, as he gives unjustified weight to preliminary information, and he does not alter the initial diagnostic assumption . . . when further contradictory evidence becomes available." Resp. Ex. C at 4.

[49] Alberto J. Espay et al., Current Concepts in Diagnosis and Treatment of Functional Neurologic Disorders, 75 JAMA Neurology 1132 (2018).

[50] Diana Kwon, A Disorder of Mind and Brain, Sci. Am., Nov. 2020, at 60.

[51] Jon Stone & Alan Carson, Functional Neurologic Disorders, 21 Continuum 818 (2015).

criticized her using a pejorative expression, and Petitioner developed leg weakness and falling, which resolved within a week. Id. The rapid resolution of leg weakness without a continued functional deficit, according to Dr. Jamieson, can occur in persons with FND. Id.

3. Respondent's Expert, Dr. Mehrdad Matloubian[52]

a. Background and Qualifications

Dr. Matloubian is a "physician-scientist with clinical training in adult rheumatology." Resp. Ex. B at 1. He is board-certified in rheumatology and internal medicine. Id.; Resp. Ex. B-1 at 1-2. He received his M.D. as well as a Ph.D in virology/immunology from University of California, Los Angeles. Resp. Ex. B at 1; Resp. Ex. B-1 at 1. Since 2001, he has taught at the University of California, San Francisco where he is now a Professor. Resp. Ex. B-1 at 2. Dr. Matloubian's research for the past 20 years has been focused on "innate and adaptive immune responses, including those of T and B cells, to acute and chronic viral infections." Resp. Ex. B at 1. He has published numerous peer-reviewed articles in these areas. Id.; Resp. Ex. B-1 at 10-15. As an immunologist and board-certified rheumatologist who actively evaluates and treats patients, Dr. Matloubian is qualified to address both diagnostic and immunological issues regarding "complex autoimmune diseases." Resp. Ex. B at 1. Dr. Matloubian is not a trained neurologist. Resp. Ex. B at 8.

b. Diagnosis Opinion

i. Transverse Myelitis

Dr. Matloubian acknowledged he is a rheumatologist, not a trained neurologist, and because of this, he deferred to Dr. Jamieson's opinions on Petitioner's presumed TM diagnosis. Resp. Ex. B at 8. However, he discussed TM, asserting "[he] [is] familiar with the types of work-up done in [neurologic] evaluations" as he encounters neurologic diseases in his practice as a rheumatologist, and he provided his opinions as to whether Petitioner had TM. Id. at 8-10.

Like Dr. Jamieson, Dr. Matloubian explained TM "is a neuroinflammatory disease of the spinal cord that typically presents with 'rapid onset of weakness, sensory alteration, and bowel or bladder dysfunction.'" Resp. Ex. B at 8-9 (quoting Resp. Ex. B-3 at 1). Diagnostic criteria for TM include "1) sensory, motor[,] or autonomic dysfunction attributable to the spinal cord; 2) T2 hyperintense signal change in the spinal cord assessed by an MRI; and 3) no evidence of a compressive lesion of the spinal cord." Id. at 9 (citing Resp. Ex. B-3 at 8). Other diagnostic criteria include bilateral signs and/or symptoms, inflammation in CSF, elevated IgG index, contrast enhancement on MRI, and clearly defined level of change in sensation. Id.

[52] Dr. Matloubian submitted one expert report. Resp. Ex. B.

For additional support, he cited Sellner et al.,[53] a nine-year retrospective study that described the spectrum of clinical presentations and neuroimaging and laboratory findings in patients with acute TM. Resp. Ex. B-4 at 1-2. Of the 63 patients in their study, all presented with sensory disturbances, 30 (47.6%) had motor system dysfunction, and 12 (19%) had autonomic dysfunction. Id. at 2. Pleocytosis was present in 29/59 patients (49.2%) and spinal cord lesions were present in 57 patients (90.4%). Id. at 3. Overall, diagnosis of TM was supported by an abnormal MRI and CSF in 52 patients (82%), with the remaining 11 patients having either a spinal cord lesion on MRI suggestive of myelitis, abnormal CSF findings, or EMG/NCS evidence of spinal cord dysfunction. Id. at 1.

Here, Petitioner presented to the ED with complaints of lower extremity weakness that progressed over two days leading to falls. Resp. Ex. B at 9. Petitioner did not report sensory changes or bladder, bowel, or autonomic dysfunction. Id. Physical examination revealed lower extremity weakness "without any definable spinal cord level of sensory change." Id. She had hyperreflexia, which Dr. Matloubian noted was "not a new finding" since she had hyperreflexia in September 2018, prior to her first HPV vaccine. Id. Petitioner's MRIs failed to show any inflammatory lesions or lesions associated with a demyelinating process. Id. And her CSF did not show inflammation. Id. Repeat diagnostic tests, including MRIs, EMG/NCS, and bloodwork, over the following months were negative. Id. Given the lack of objective findings, Dr. Matloubian opined "Petitioner does not fit the diagnostic criteria for TM." Id. And her rapid recovery within three days "[was] quite unusual" for TM "since it typically takes several months to sometimes years for individuals to recover." Id. (citing Resp. Ex. B-3 at 19).

He opined Petitioner's continued complaints of headaches, back pain, shooting pain in her leg, and tingling in her left arm and leg were not related to her presumed TM. Resp. Ex. B at 9. Petitioner complained of headaches/migraines prior to vaccination, and thus, it was "not a manifestation of her presumed TM." Id. Her back pain was documented to be due to musculoskeletal causes (paraspinal muscle tenderness on examination), not neurologic. Id. (citing Pet. Ex. 4 at 218). Complaints of shooting pain were not corroborated by findings seen on MRIs or EMG/NCS. Id. And tingling in her left arm and leg, according to Dr. Matloubian, "would be suggestive of an incomplete lesion higher in the spinal cord, and also quite different from the bilateral leg weakness she initially presented with." Id. at 10.

Overall, given Petitioner's initial presentation, normal diagnostic tests, and rapid response to high dose steroids with complete recovery within days, all of which is "highly atypical for TM," Dr. Matloubian opined Petitioner did not have TM. Resp. Ex. B at 10. Her subsequent complaints were ruled out as a CNS process, including TM, and thereafter removed from her problem list by her neurologist. Id.

ii. Fibromyalgia

Two years after vaccination, in April 2021, at an appointment with rheumatologist Dr. Neal, Petitioner was diagnosed with fibromyalgia after complaining of diffuse pain. Resp. Ex. B

[53] J. Sellner et al., Diagnostic Workup of Patients with Acute Transverse Myelitis: Spectrum of Clinical Presentation, Neuroimaging and Laboratory Findings, 47 Spinal Cord 312 (2009).

at 10. Dr. Matloubian opined that Petitioner's symptoms of paresthesias, back pain, diffuse tenderness can be attributed to fibromyalgia, and many of these symptoms were present prior to Petitioner's HPV vaccinations. Id. at 10-11.

Dr. Matloubian does not assert the HPV vaccine caused or contributed to Petitioner's fibromyalgia.[54] See Resp. Ex. B.

c. Causation Opinion

Dr. Matloubian opined that Petitioner's alleged vaccine-related injury of TM was not caused by her June 7, 2019 HPV vaccination. Resp. Ex. B at 22.

i. Althen Prong One

Dr. Matloubian opined that Dr. Steinman failed to provide preponderant evidence to support his theory. Resp. Ex. B at 22.

Dr. Matloubian began with a general summary of TM, describing it as "a mixed inflammatory disorder that affect neurons, axons, and oligodendrocytes and myelin." Resp. Ex. B at 11-12 (quoting Resp. Ex. B-3 at 2). "Although immune-mediated damage through molecular mimicry has been suggested, . . . this has not been established for any viral pathogen in postinfectious myelopathy." Id. at 11.

He noted there are various potential theories that have been posited in literature to explain how viruses, including EBV, could lead to TM. Resp. Ex. B at 11-12 (citing Resp. Ex. B-3 at 3). Because various viruses/infections are associated with TM, Dr. Matloubian opined "it is not surprising for viruses to cause neurological disease through direct infection or the immune response to the viral antigens." Id. at 11. Additionally, TM can be associated with autoimmune diseases and vascular etiologies, and TM can be idiopathic. Id.

Dr. Matloubian opined there was no literature to support a link between HPV infection and development of demyelinating diseases, like TM. Resp. Ex. B at 12. He cited the 2012 IOM Report, which found no epidemiologic evidence to assess the risk of TM following HPV vaccination and concluded "[t]he evidence [was] inadequate to accept or reject a causal relationship between HPV vaccine and [TM]." Id. (citing Pet. Ex. 28 at 537).

Following the publication of this IOM report, several controlled studies assessed the risk of autoimmune and inflammatory diseases following HPV vaccination. Resp. Ex. B at 12. Chao

[54] For Dr. Matloubian's discussion on fibromyalgia and why he opined Petitioner has fibromyalgia, see Resp. Ex. B at 10-11.

et al.,[55] published in 2012, conducted a safety study of the quadrivalent HPV vaccine[56] in women from 2006 to 2010 to monitor development of autoimmune conditions. Resp. Ex. B-10 at 1-2. Among the 189,629 women in the study, 1,014 had potential new-onset autoimmune disease. Id. at 4. Twenty-one were confirmed to have new onset neurological conditions with diagnostic certainty. Id. Of the 21, 19 had disease onset after the first HPV dose, with a median onset of 46.5 days (range: 1-161 days). Id. No increased risk of autoimmune diseases, including "other demyelinating diseases of the [CNS]," was found within 180 days after HPV vaccination. Id. at 1-2, 9. The study did not specifically mention or discuss TM, or indicate whether TM was included in "other demyelinating diseases of the [CNS]." See id.

In 2015, Scheller et al.[57] published a study that examined the risk of demyelinating diseases of the CNS (including TM) in females (aged 10-44 years) in Demark and Sweden from 2006 to 2013 who received the quadrivalent HPV vaccination.[58] Resp. Ex. B-9 at 1. The authors found 73 cases of MS and 90 cases of other demyelinating diseases occurred during the vaccinated risk period (two years). Id. at 3. They concluded there was no increased risk of MS or other demyelinating diseases following HPV vaccination. Id. at 4.

Dr. Matloubian also cited two studies from Grimaldi-Bensouda et al. that assessed the risk of autoimmune disorders following HPV vaccinations. Resp. Ex. B-11;[59] Resp. Ex. B-12.[60] The first, published in 2013, focused on females, aged 14-26 years, from December 2007 to April 2011 and whether the quadrivalent HPV vaccine was associated with six autoimmune disorders, including "central demyelination and [MS]."[61] Resp. Ex. B-11 at 1-2. Similarly, the second study from Grimaldi-Bensouda et al., published in 2016, assessed the risk of autoimmune disorders (including central demyelination/MS) associated with the HPV vaccine in females

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[55] C. Chao et al., Surveillance of Autoimmune Conditions Following Routine Use of Quadrivalent Human Papillomavirus Vaccine, 271 J. Internal Med. 193 (2012).

[56] This HPV vaccine contains HPV Type 11. Resp. Ex. B-10 at 1.

[57] Nikolai Madrid Scheller et al., Quadrivalent HPV Vaccination and Risk of Multiple Sclerosis and Other Demyelinating Diseases of the Central Nervous System, 313 JAMA 54 (2015).

[58] This HPV vaccine also contains HPV Type 11. Resp. Ex. B at 19; Resp. Ex. B-9 at 2.

[59] L. Grimaldi-Bensouda et al., Autoimmune Disorders and Quadrivalent Human Papillomavirus Vaccination of Young Female Subjects, 275 J. Internal Med. 398 (2014). This HPV vaccine contains HPV Type 11. Resp. Ex. B-11 at 2; see also Resp. Ex. B-12 at 2 (noting Resp. Ex. B-11 used the quadrivalent HPV vaccine with HPV Type 11).

[60] Lamiae Grimaldi-Bensouda et al., Risk of Autoimmune Diseases and Human Papilloma Virus (HPV) Vaccines: Six Years of Case Referent Surveillance, 79 J. Autoimmunity 84 (2017). Of the two HPV vaccines available in France during this study, most study participants (95.3%) received the HPV vaccine containing HPV Type 11. Resp. Ex. B-12 at 3, 4 tbl.1.

[61] It is not clear whether TM was included in this group.

(aged 11-25 years) from 2008 to 2014. Resp. Ex. B-12 at 2. The latter study followed more than double the participants over a longer period of time (6.5 years versus 3.4 years). Id. at 4. Both studies found no evidence of an increased risk of central demyelination/MS following HPV vaccination. Id.; Resp. Ex. B-11 at 5, 8.

Next, Dr. Matloubian responded to Dr. Steinman's causal theory. Resp. Ex. B at 14-22. Molecular mimicry, according to Dr. Matloubian, requires "the same T cell with its unique T cell receptor [] to see either a foreign peptide or a self-antigen peptide in the context of that person's MHC/HLA molecule." Id. at 15.

First, Dr. Matloubian took issue with Dr. Steinman's reliance on MBP as it "has not been established as a generally accepted target of autoimmunity in TM." Resp. Ex. B at 14. Dr. Matloubian noted Dr. Steinman's only article to support his contention that MBP is the relevant host target[62] for TM is Abramsky and Teitelbaum. Id. (citing Pet. Ex. 13). Dr. Matloubian found the lack of follow-up studies to confirm findings in this older 1977 article "raises questions regarding the validity and generalizability of their findings to individuals with TM." Id. More recent articles, according to Dr. Matloubian, discuss other host targets in the context of TM (e.g. MOG and AQP4), not MBP. Id. (citing Resp. Ex. B-3 at 12; Resp. Ex. B-13 at 10;[63] Resp. Ex. B-14 at 14-15, 17).[64] Thus, he opined that "[t]he absence of MBP in literature pertaining to pathogenesis of TM makes [it] highly unlikely to be relevant to this disease." Id.

Second, Dr. Matloubian opined "sequence homology searches are not sufficient to establish molecular mimicry for T cells." Resp. Ex. B at 15. And thus, Dr. Steinman's "weak sequence homology" is not evidence for molecular mimicry. Id. For support, he quoted a passage from the 2012 IOM report:

> Linear amino acid sequence homology or even similar conformational structure between an exogenous agent and a self-antigen alone are not sufficient to prove that molecular mimicry is the pathogenic mechanism for a disease. Many such homologies exist, and the vast majority of these are not associated with biologically relevant autoimmune phenomena or actual human disease.

Id. (quoting Pet. Ex. 28 at 99).

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[62] For clarity and uniformity, the undersigned refers to MBP as the "host target" rather than the "antigen" at play with TM.

[63] Bruce A.C. Cree, Acute Inflammatory Myelopathies, 122 Handbook Clinical Neurology 613 (2014).

[64] Eoin P. Flanagan, Autoimmune Myelopathies, 133 Handbook Clinical Neurology 327 (2016).

As to Dr. Steinman's sequence homology specifically, Dr. Matloubian further opined the literature does not support mimicry between the L1 peptide of HPV Type 11 and MBP.[65] Resp. Ex. B at 16-17. Nor did Dr. Matloubian's Immune Epitope Database ("IEDB")[66] search using Dr. Steinman's sequences find any exact matches to support molecular mimicry. Id. at 17. He concluded that the mimic Dr. Steinman posited here has not been shown to be recognized by human T cells and is therefore unlikely to be implicated. Id.

Further, Dr. Matloubian asserted that homology of five amino acids is not sufficient. Resp. Ex. B at 17 (citing Pet. Exs. 21-23). A finding that "only a few shared amino acids in a sequence [is] sufficient for cross reactive immunity of T cells through molecular mimicry[] is neither scientifically correct, nor generalizable, nor a true representation of the conclusions of the authors of the referenced studies." Id. For support, he cited a study that showed altering one single amino acid reduced the ability for a peptide to activate T cells. Id. at 18 (citing Resp. Ex. B-16).[67] Dr. Matloubian asserted that although there are many examples of sequence homology, most do not result in molecular mimicry and thus, it is incorrect to say "every sequence homology implies molecular mimicry." Id.

He concluded that HPV vaccination cannot cause TM via Dr. Steinman's proposed theory because (1) literature does not show MBP is the relevant self-antigen in TM; (2) the sequence homology alleged was not supported by the literature or IEDB; and (3) studies have not found an association between HPV vaccination and TM. Resp. Ex. B at 22.

ii. Althen Prong Two

Dr. Matloubian opined that "Petitioner's reported symptoms of weakness were biologically unrelated to her [June 7, 2019] HPV immunization." Resp. Ex. B at 13. Petitioner did not have many features of TM, including an abnormal MRI or inflammatory findings in her CSF. Id. Her symptoms were entirely motor and resolved days after steroid treatment. Id. Within one month of vaccination, her leg weakness completely resolved. Id. Extensive neurologic work up did not show evidence of TM, and she was eventually diagnosed with fibromyalgia, "which is not an inflammatory or autoimmune process and can be associated with her pre-existing conditions." Id. Petitioner had evidence of recent EBV infection, "which has been associated with demyelinating diseases of CNS, including TM." Id. And multiple

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[65] Dr. Matloubian explained that he searched the database of 4,824 peptides identified by the authors in Birnbaum et al. and determined the L1 peptide of HPV Type 11 or even portions of the peptide were not included in the database. Resp. Ex. B at 17 (citing Pet. Ex. 17; Resp. Ex. B-19). Thus, Dr. Matloubian concluded Dr. Steinman was incorrect. Id.

[66] The IEDB "catalogs experimental data on antibody and T cell epitopes studied in humans and other animal species in the context of infectious disease, allergy, autoimmunity[,] and transplantation." Immune Epitope Database & Tools, https://www.iedb.org/ (last updated Mar. 9, 2025).

[67] Mark A. Daniels, Thymic Selection Threshold Defined by Compartmentalization of Ras/MAPK Signaling, 444 Nature 724 (2006).

epidemiologic studies have not found increased risk of autoimmune diseases following HPV vaccination. Id.

Dr. Matloubian opined that if Petitioner did have TM, EBV is "an established cause of TM" and "should be considered[] [] since prior infection with [EBV] has recently been epidemiologically associated with an increased risk of [MS], a demyelinating disease of CNS." Resp. Ex. B at 12 (citing Resp. Ex. B-7 at 1);[68] see also, e.g., Resp. Ex. B-3 at 30, 33 (noting EBV is associated with TM). Petitioner tested positive for antibodies to EBV early antigen D, seen during a recent infection, and anti-ENBA antibodies, which ruled out an ongoing infection. Resp. Ex. B at 11 (citing Pet. Ex. 2 at 30); see also Resp. Ex. B-2 at 13 (noting early antigen D antibodies are "consistent with recent infection" and EBNA antibodies are "expressed only when the virus begins to establish latency").[69] He opined that "[t]ogether these two positive blood tests are consistent with a recent but not currently active EBV infection." Resp. Ex. B at 11.

Further, Dr. Matloubian asserted Dr. Steinman did not explain why the HPV vaccine "was more likely than [P]etitioner's documented EBV infection to have caused her alleged TM" when "[t]here is epidemiologic evidence suggestive of a link between EBV infection and MS" and no link between HPV vaccination and neuroinflammatory diseases like MS. Resp. Ex. B at 14.

He also took issue with Dr. Steinman's sequence homology because "without knowing what MHC/HLA molecules [P]etitioner has, there is no way . . . to predict which peptides will potentially be seen by her T cells. Therefore, there is no basis for a theory that molecular mimicry occurs at the T cell level in this particular case." Resp. Ex. B at 16 (citing Resp. Ex. B-15 (noting MHC/HLA molecules are different in every individual and the MHC/HLA molecules determines what peptides present to T cells)).[70] Additionally, because molecular mimicry between L1 protein of HPV Type 11 and MBP was not supported by the literature and IEDB, then it is "very unlikely" for this to have occurred in Petitioner. Id. at 17.

iii. Althen Prong Three

As to timing, Dr. Matloubian cited studies that have analyzed the timing of TM and other similar conditions following HPV vaccination. Resp. Ex. B at 19. Scheller et al., for example, did not find an increased risk of neuroinflammatory diseases for up to two years following HPV vaccination. Id. (citing Resp. Ex. B-9 at 1, 4-5). Dr. Matloubian noted the HPV vaccine in Scheller et al. also contained the L1 protein of HPV Type 11, Dr. Steinman's proposed protein

[68] Kjetil Bjornevik et al., Longitudinal Analysis Reveals High Prevalence of Epstein-Barr Virus Associated with Multiple Sclerosis, 375 Science 296 (2022).

[69] Mark D. Aronson et al., Infectious Mononucleosis, UpToDate, https://www.uptodate.com/contents/infectious-mononucleosis (last updated Mar. 19, 2021).

[70] Antigen Presentation to T Lymphocytes and the Functions of Major Histocompatibility Complex Molecules, in Cellular and Molecular Immunology 117 (Abul K. Abbas et al. eds., 9th ed. 2018).

involved here. Id. He opined that Scheller et al. "argues strongly against Dr. Steinman's molecular mimicry theory in this case" since an association was not shown in Scheller et al. Id.

Dr. Matloubian also took issue with Dr. Steinman's reliance on Lai et al., as it was not representative of what occurs in an individual after vaccination in order for a primary B or T cell immune response or recall response to occur. Resp. Ex. B at 20 (citing Pet. Ex. 35). Dr. Matloubian cited a text from Abbas et al. that depicted the steps that must occur for activation of T cells following vaccination, showing neurological symptoms of weakness characteristic of TM could not occur within the day or two alleged by Dr. Steinman even with a recall response. Id. at 21-22 (citing Resp. Ex. B-15).

He concluded that Petitioner's HPV vaccine administered on June 7, 2019 was not the "more likely than not" cause of her alleged TM because (1) there is not preponderant evidence of an association between HPV vaccination and TM; (2) the theory alleged could not have occurred in Petitioner; (3) she had a recent EBV infection, which is a known cause of TM; and (4) her rapid onset is inconsistent with a recall response. Resp. Ex. B at 22.

III. DISCUSSION

A. Standards for Adjudication

The Vaccine Act was established to compensate vaccine-related injuries and deaths. § 10(a). "Congress designed the Vaccine Program to supplement the state law civil tort system as a simple, fair and expeditious means for compensating vaccine-related injured persons. The Program was established to award 'vaccine-injured persons quickly, easily, and with certainty and generosity.'" Rooks v. Sec'y of Health & Hum. Servs., 35 Fed. Cl. 1, 7 (1996) (quoting H.R. Rep. No. 908 at 3, reprinted in 1986 U.S.C.C.A.N. at 6287, 6344).

Petitioner's burden of proof is by a preponderance of the evidence. § 13(a)(1). The preponderance standard requires a petitioner to demonstrate that it is more likely than not that the vaccine at issue caused the injury. Moberly v. Sec'y of Health & Hum. Servs., 592 F.3d 1315, 1322 n.2 (Fed. Cir. 2010). Proof of medical certainty is not required. Bunting v. Sec'y of Health & Hum. Servs., 931 F.2d 867, 873 (Fed. Cir. 1991). Petitioner need not make a specific type of evidentiary showing, i.e., "epidemiologic studies, rechallenge, the presence of pathological markers or genetic predisposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect." Capizzano v. Sec'y of Health & Hum. Servs., 440 F.3d 1317, 1325 (Fed. Cir. 2006). Instead, Petitioner may satisfy her burden by presenting circumstantial evidence and reliable medical opinions. Id. at 1325-26.

In particular, Petitioner must prove that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." Moberly, 592 F.3d at 1321 (quoting Shyface v. Sec'y of Health & Hum. Servs., 165 F.3d 1344, 1352-53 (Fed. Cir. 1999)); see also Pafford v. Sec'y of Health & Hum. Servs., 451 F.3d 1352, 1355 (Fed. Cir. 2006). The received vaccine, however, need not be the predominant cause of the injury. Shyface, 165 F.3d at 1351. A petitioner who satisfies this burden is entitled to compensation unless Respondent can prove, by a preponderance of the evidence, that the vaccinee's injury is "due to factors

unrelated to the administration of the vaccine." § 13(a)(1)(B). However, if a petitioner fails to establish a prima facie case, the burden does not shift. Bradley v. Sec'y of Health & Hum. Servs., 991 F.2d 1570, 1575 (Fed. Cir. 1993).

"Regardless of whether the burden ever shifts to the [R]espondent, the special master may consider the evidence presented by the [R]espondent in determining whether the [P]etitioner has established a prima facie case." Flores v. Sec'y of Health & Hum. Servs., 115 Fed. Cl. 157, 162-63 (2014); see also Stone v. Sec'y of Health & Hum. Servs., 676 F.3d 1373, 1379 (Fed. Cir. 2012) ("[E]vidence of other possible sources of injury can be relevant not only to the 'factors unrelated' defense, but also to whether a prima facie showing has been made that the vaccine was a substantial factor in causing the injury in question."); de Bazan v. Sec'y of Health & Hum. Servs., 539 F.3d 1347, 1353 (Fed. Cir. 2008) ("The government, like any defendant, is permitted to offer evidence to demonstrate the inadequacy of the [P]etitioner's evidence on a requisite element of the [P]etitioner's case-in-chief."); Pafford, 451 F.3d at 1358-59 ("[T]he presence of multiple potential causative agents makes it difficult to attribute 'but for' causation to the vaccination. . . . [T]he Special Master properly introduced the presence of the other unrelated contemporaneous events as just as likely to have been the triggering event as the vaccinations.").

B. Factual Issues

Petitioner must prove, by a preponderance of the evidence, the factual circumstances surrounding her claim. § 13(a)(1)(A). To resolve factual issues, the special master must weigh the evidence presented, which may include contemporaneous medical records and testimony. See Burns v. Sec'y of Health & Hum. Servs., 3 F.3d 415, 417 (Fed. Cir. 1993) (explaining that a special master must decide what weight to give evidence including oral testimony and contemporaneous medical records). Contemporaneous medical records, "in general, warrant consideration as trustworthy evidence." Cucuras v. Sec'y of Health & Hum. Servs., 993 F.2d 1525, 1528 (Fed. Cir. 1993). But see Kirby v. Sec'y of Health & Hum. Servs., 997 F.3d 1378, 1382 (Fed. Cir. 2021) (rejecting the presumption that "medical records are accurate and complete as to all the patient's physical conditions"); Shapiro v. Sec'y of Health & Hum. Servs., 101 Fed. Cl. 532, 538 (2011) ("[T]he absence of a reference to a condition or circumstance is much less significant than a reference which negates the existence of the condition or circumstance." (quoting Murphy v. Sec'y of Health & Hum. Servs., 57 Cl. Ct. 726, 733 (1991), aff'd per curiam, 968 F.2d 1226 (Fed. Cir. 1992))), recons. den'd after remand, 105 Fed. Cl. 353 (2012), aff'd mem., 503 F. App'x 952 (Fed. Cir. 2013).

There are situations in which compelling testimony may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. Campbell ex rel. Campbell v. Sec'y of Health & Hum. Servs., 69 Fed. Cl. 775, 779 (2006) ("[L]ike any norm based upon common sense and experience, this rule should not be treated as an absolute and must yield where the factual predicates for its application are weak or lacking."); Lowrie v. Sec'y of Health & Hum. Servs., No. 03-1585V, 2005 WL 6117475, at *19 (Fed. Cl. Spec. Mstr. Dec. 12, 2005) ("[W]ritten records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent." (quoting Murphy, 57 Cl. Ct. at 733)). Ultimately, a determination regarding a witness's credibility is needed when determining the

weight that such testimony should be afforded. Andreu v. Sec'y of Health & Hum. Servs., 569 F.3d 1367, 1379 (Fed. Cir. 2009); Bradley, 991 F.2d at 1575.

Despite the weight afforded to medical records, special masters are not bound rigidly by those records in determining onset of a petitioner's symptoms. Valenzuela v. Sec'y of Health & Hum. Servs., No. 90-1002V, 1991 WL 182241, at *3 (Fed. Cl. Spec. Mstr. Aug. 30, 1991); see also Eng v. Sec'y of Health & Hum. Servs., No. 90-1754V, 1994 WL 67704, at *3 (Fed. Cl. Spec. Mstr. Feb. 18, 1994) (Section 13(b)(2) "must be construed so as to give effect also to § 13(b)(1) which directs the special master or court to consider the medical records (reports, diagnosis, conclusions, medical judgment, test reports, etc.), but does not require the special master or court to be bound by them").

C. Causation

To receive compensation through the Program, Petitioner must prove either (1) that she suffered a "Table Injury"—i.e., an injury listed on the Vaccine Injury Table—corresponding to a vaccine that she received, or (2) that she suffered an injury that was actually caused by a vaccination. See §§ 11(c)(1), 13(a)(1)(A); Capizzano, 440 F.3d at 1319-20. Petitioner must show that the vaccine was "not only a but-for cause of the injury but also a substantial factor in bringing about the injury." Moberly, 592 F.3d at 1321 (quoting Shyface, 165 F.3d at 1352-53).

Because Petitioner does not allege she suffered a Table Injury, she must prove a vaccine she received actually caused her injury. To do so, Petitioner must establish, by preponderant evidence: "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." Althen, 418 F.3d at 1278.

The causation theory must relate to the injury alleged. Petitioner must provide a sound and reliable medical or scientific explanation that pertains specifically to this case, although the explanation need only be "legally probable, not medically or scientifically certain." Knudsen v. Sec'y of Health & Hum. Servs., 35 F.3d 543, 548-49 (Fed. Cir. 1994). Petitioner cannot establish entitlement to compensation based solely on her assertions; rather, a vaccine claim must be supported either by medical records or by the opinion of a medical doctor. § 13(a)(1). In determining whether Petitioner is entitled to compensation, the special master shall consider all material in the record, including "any . . . conclusion, [or] medical judgment . . . which is contained in the record regarding . . . causation." § 13(b)(1)(A). The special master must weigh the submitted evidence and the testimony of the parties' proffered experts and rule in Petitioner's favor when the evidence weighs in her favor. See Moberly, 592 F.3d at 1325-26 ("Finders of fact are entitled—indeed, expected—to make determinations as to the reliability of the evidence presented to them and, if appropriate, as to the credibility of the persons presenting that evidence."); Althen, 418 F.3d at 1280 (noting that "close calls" are resolved in Petitioner's favor).

Testimony that merely expresses the possibility—not the probability—is insufficient, by itself, to substantiate a claim that such an injury occurred. See Waterman v. Sec'y of Health &

Hum. Servs., 123 Fed. Cl. 564, 573-74 (2015) (denying Petitioner's motion for review and noting that a possible causal link was not sufficient to meet the preponderance standard). The Federal Circuit has made clear that the mere possibility of a link between a vaccination and a petitioner's injury is not sufficient to satisfy the preponderance standard. Moberly, 592 F.3d at 1322 (emphasizing that "proof of a 'plausible' or 'possible' causal link between the vaccine and the injury" does not equate to proof of causation by a preponderance of the evidence); Boatmon, 941 F.3d at 1359-60. While certainty is by no means required, a possible mechanism does not rise to the level of preponderance. Moberly, 592 F.3d at 1322; see also de Bazan, 539 F.3d at 1351.

IV. ANALYSIS

A. Diagnosis

As Federal Circuit precedent establishes, in certain cases it is appropriate to determine the nature of an injury before engaging in the Althen analysis. Broekelschen v. Sec'y of Health & Hum. Servs., 618 F.3d 1339, 1346 (Fed. Cir. 2010). Since "each prong of the Althen test is decided relative to the injury [,]" determining facts relating to the claimed injury can be significant. Id. Here, the parties disagree as to Petitioner's diagnosis.

For the following reasons, the undersigned finds Petitioner's appropriate diagnosis is TM.

First, numerous treating physicians diagnosed Petitioner with TM. During Petitioner's hospitalization, at least four different treating physicians diagnosed Petitioner with TM. ED physician Dr. Ruschman noted that "[s]he could have something like [TM]." Pet. Ex. 4 at 314. Dr. Opii noted she was admitted for "suspected [TM]." Id. at 318. Neurologist Dr. Sheffield's assessment was "[m]yelopathy—likely [TM]." Id. at 321. And the following day, her assessment was TM. Id. at 346. Hospitalist Dr. Woody assessed Petitioner with "[p]resumed [a]cute [TM]." Id. at 342, 345. Petitioner was discharged with a diagnosis of "[p]resumed [a]cute [TM]." Id. at 322. And following Petitioner's hospitalization, Petitioner's treating neurologist, Dr. Winkley, noted Petitioner "had band like sensation [at T10] with increased sensitivity." Pet. Ex. 5 at 189. He diagnosed Petitioner with "[p]resumed [a]cute [TM]" with "[symptoms] of presumed T10 [TM]." Id. at 197. Here, the undersigned gives weight to the opinions of Petitioner's treating physicians as they are "in the best position" to determine Petitioner's injury. See Andreu, 569 F.3d at 1367; Capizzano, 440 F.3d at 1326; Cucuras, 993 F.2d at 1528 (noting contemporaneous medical records, "in general, warrant consideration as trustworthy evidence").

Second, although Petitioner's symptoms were not "typical" of TM and diagnostic testing did not confirm a diagnosis of TM, both parties filed literature confirming symptomology and diagnostic testing results vary in patients with TM. The TM Consortium Working Group, for example, noted "[t]here is often a clearly defined [level] of sensory dysfunction, and spinal MRI and lumbar puncture often show evidence of acute inflammation." Resp. Ex. A-3 at 1 (emphasis added). At maximal deficit, "approximately 50% of patients have lost all movements of their legs, virtually all patients have bladder dysfunction, and 80 to 94% of patients have numbness, paresthesias, or band-like dysesthesias." Id. Similarly, a study from Sellner et al. examined 63

patients with acute TM and found all presented with sensory disturbances, 30 (47.6%) had motor system dysfunction, and 12 (19%) had autonomic dysfunction. Resp. Ex. B-4 at 2. Pleocytosis was present in 29/59 patients (49.2%) and spinal cord lesions were present in 57 patients (90.4%). Id. at 3. Thus, the "typical" symptoms and diagnostic evidence of inflammation seen in TM are not present in all TM patients. In fact, "a significant percentage of individuals with a clinical pattern that otherwise resembles TM do not meet the inflammatory features; therefore, the absence of inflammatory markers does not rule out TM." Pet. Ex. 34 at 8. "Clinical events that are consistent with [TM] but that are not associated with [CSF] abnormalities or abnormalities detected on MRI and that have no identifiable underlying cause are categorized as possible idiopathic [TM]." Resp. Ex. A-6 at 2.

Third, Petitioner was treated for TM with high-dose intravenous glucocorticoids, and thereafter recovered, consistent with what is often seen in patients with TM. See Pet. Ex. 34 at 15-16; Resp. Ex. A-6 at 5.

Next, the undersigned finds Petitioner did not have an FND. None of Petitioner's treating physicians diagnosed her with a functional disorder. Nor did any treating physician mention FND in their records or include such diagnosis in their differentials. This diagnosis was introduced only by Dr. Jamieson, years after onset, and on behalf of the Respondent in this matter. The undersigned finds Petitioner's treating physician's opinions more persuasive given their first-hand knowledge treating Petitioner in 2019.

Lastly, the undersigned finds a diagnosis of fibromyalgia in April 2021, almost two years after the subject HPV vaccination, to be too remote in time to explain Petitioner's symptoms beginning in June 2019. The undersigned also finds a diagnosis of fibromyalgia does not fit with Petitioner's earlier clinical course in June 2019, consisting of leg weakness, difficulty ambulating, and multiple falls, and the treating physicians' diagnosis of TM.

Overall, the undersigned finds, by a preponderance of evidence, Petitioner's accurate diagnosis is TM.

B. Causation

1. Althen Prong One

Under Althen prong one, Petitioner must set forth a medical theory explaining how the received vaccine could have caused the sustained injury. Andreu, 569 F.3d at 1375; Pafford, 451 F.3d at 1355-56. Petitioner's theory of causation need not be medically or scientifically certain, but it must be informed by a "sound and reliable" medical or scientific explanation. Boatmon, 941 F.3d at 1359; see also Knudsen, 35 F.3d at 548; Veryzer v. Sec'y of Health & Hum. Servs., 98 Fed. Cl. 214, 257 (2011) (noting that special masters are bound by both § 13(b)(1) and Vaccine Rule 8(b)(1) to consider only evidence that is both "relevant" and "reliable"). If Petitioner relies upon a medical opinion to support her theory, the basis for the opinion and the reliability of that basis must be considered in the determination of how much weight to afford the offered opinion. See Broekelschen, 618 F.3d at 1347 ("The special master's decision often times is based on the credibility of the experts and the relative persuasiveness of their competing

theories."); Perreira v. Sec'y of Health & Hum. Servs., 33 F.3d 1375, 1377 n.6 (Fed. Cir. 1994) (stating that an "expert opinion is no better than the soundness of the reasons supporting it" (citing Fehrs v. United States, 620 F.2d 255, 265 (Ct. Cl. 1980))).

The undersigned finds Petitioner failed to provide preponderant evidence of a sound and reliable theory to explain how the HPV vaccine can cause TM for the following reasons.

First, Petitioner's proposed mechanism of molecular mimicry based on homology between the L1 protein of HPV Type 11 and MBP falls short and fails to meet the level of preponderance of the evidence.

Dr. Steinman, relying on Abramsky and Teitelbaum, chose to focus on MBP "because it is the most abundant myelin protein" and "was a target of the immune attack in acute [TM]" in Abramsky and Teitelbaum. Pet. Ex. 11 at 5. Dr. Steinman provided no other evidence to support his assertion that MBP is the target in TM. In fact, he notes his theory is based on mimicry with "proteins known to be shared with neuronal antigens attacked by the human immune system in polyneuropathy." Id. at 23; Pet. Ex. 32 at 6. He failed to explain how a protein involved with neuropathy in the peripheral nerves can be relevant to TM, a CNS disorder. And, as both Dr. Steinman and Dr. Matloubian acknowledged, more recent literature discusses other host targets (MOG and AQP4) at play in TM. The undersigned finds Dr. Steinman failed to sufficiently acknowledge this in his reports.

Even assuming Dr. Steinman is correct that MBP is a target in TM, he fails to provide preponderant evidence of homology between MBP and L1 protein of HPV Type 11, or that such homology can trigger molecular mimicry and lead to TM. To support his opinion that there is sequence homology between MBP and the L1 protein for HPV Type 11, he noted there were five identical amino acids, three of which were consecutive at "at critical binding points." Pet. Ex. 11 at 9-10. Dr. Steinman did not explain the significance of "critical binding points," or any effect this may have had so as to trigger molecular mimicry.

He cites to a study from Wucherpfennig and Strominger to show sequence similarity between the live virus HPV and MBP. However, this sequence similarity was with the L2 protein of HPV Type 7, not the L1 protein of HPV Type 11. Dr. Steinman failed to address this discrepancy, or provide any evidence to support a finding of an association between the L2 protein of HPV Type 7 and the L1 protein of HPV Type 11. As such, the undersigned does not find this study informative.

In past cases, Dr. Steinman has conducted a BLAST[71] search to determine whether a component of the vaccine at issue has alignment with human proteins that are known to be attacked based on scientific literature. In these instances, he has shown the steps taken to conduct the BLAST search in his expert reports to verify sequence homology. He did not

[71] A BLAST (Basic Local Alignment Search Tool) search "finds regions of similarity between biological sequences. The program compares nucleotide or protein sequences to sequence databases and calculates the statistical significance." BLAST, https://blast.ncbi.nlm.nih.gov/Blast.cgi (last visited Mar. 25. 2025).

provide such foundational evidence in this case. See, e.g., Juranek v. Sec'y of Health & Hum. Servs., No. 19-226V, 2025 WL 399501, at *16-17 (Fed. Cl. Spec. Mstr. Jan. 8, 2025); Simeneta v. Sec'y of Health & Hum. Servs., No. 18-859V, 2024 WL 4881411, at *15-16 (Fed. Cl. Spec. Mstr. Oct. 31, 2024); Mullins ex rel. K.M. v. Sec'y of Health & Hum. Servs., No. 19-320V, 2024 WL 4045424, at *16 (Fed. Cl. Spec. Mstr. Aug. 8, 2024).

Additionally, Dr. Steinman, in other cases, had confirmed the relevance of the sequence by conducting an IEDB search of peer reviewed literature to determine whether the sequence has been studied or referenced in the literature. See, e.g., Juranek, 2025 WL 399501, at *16-17; Simeneta, 2024 WL 4881411, at *15 n.52; Mullins, 2024 WL 4045424, at *17. Here, Dr. Steinman did not conduct an IEDB search. Respondent's expert Dr. Matloubian searched the IEDB and did not find matches. Here Dr. Steinman did not show his BLAST search, he did not identify peer-reviewed medical literature that referenced the purported peptide sequences he identified, and the IEDB did not identify any data to support homology based on the sequences.

In contrast, Respondent, via Dr. Matloubian, provided studies that examined the risk of CNS diseases, including TM, following HPV vaccinations, some of which were confirmed to contain HPV Type 11, and these studies do not show a causal association. Chao et al. (2012) determined there was no increased risk of "demyelinating diseases of the [CNS]" following HPV vaccination, a vaccine that included HPV Type 11. Resp. Ex. B-10 at 1-2, 9. Like Chao et al., Scheller et al. (2015) examined the risk of demyelinating diseases of the CNS, including TM, following HPV vaccination, which included HPV Type 11, and found no increased risk of MS or other demyelinating diseases following HPV vaccination. Resp. Ex. B-9 at 1, 4. And the 2014 and 2017 studies from Grimaldi-Bensouda et al., one of which included HPV vaccine with HPV Type 11 and the other had 95.3% of participants who received the HPV vaccine containing HPV Type 11, also found no evidence of an increased risk of central demyelination/MS following HPV vaccination. Resp. Ex. B-11 at 2, 5, 8; Resp. Ex. B-12 at 3-4. Dr. Steinman did not acknowledge, address, or discuss these studies in his reports.

Although a petitioner need not make a specific type of evidential showing (i.e., epidemiologic studies) to satisfy her burden, special masters shall still consider and weigh the evidence in the record, including the epidemiological studies filed. See § 13(b)(1) (indicating the special master shall consider all materials in the record); Capizzano, 440 F.3d at 1325-26; Grant v. Sec'y of Health & Hum. Servs., 956 F.2d 1144, 1149 (Fed. Cir. 1992) (finding "epidemiological studies are probative medical evidence relevant to causation" and "considerable weight [is] due to epidemiological studies in the absence of direct evidence of actual causation"); see also Moberly, 592 F.3d at 1325-26 ("Finders of fact are entitled—indeed, expected—to make determinations as to the reliability of the evidence presented to them and, if appropriate, as to the credibility of the persons presenting that evidence.").

Overall, the undersigned finds Dr. Steinman's theory does not meet the preponderance standard. His theory lacks sufficient supportive evidence, and is overall not sound and reliable. The undersigned will not rely on "opinion evidence that is connected to existing data only by the ipse dixit of the expert." Prokopeas v. Sec'y of Health & Hum. Servs., No. 04-1717V, 2019 WL 2509626, at *19 (Fed. Cl. Spec. Mstr. May 24, 2019) (quoting Moberly, 592 F.3d at 1315). "[C]onclusory expert statements that are not themselves backed up with reliable scientific

support" are consistently rejected. Kreizenbeck v. Sec'y of Health & Hum. Servs., No. 08-209V, 2018 WL 3679843, at *31 (Fed. Cl. Spec. Mstr. June 22, 2018), mot. for rev. den'd, 141 Fed. Cl. 138, aff'd, 945 F.3d 1362 (Fed. Cir. 2020).

Lastly, although rulings and decisions by other special masters are not binding on the undersigned, the undersigned notes there is one reasoned decision involving the HPV vaccine and TM. See White v. Sec'y of Health & Hum. Servs., No. 15-1521V, 2019 WL 7563239, at *21-24 (Fed. Cl. Spec. Mstr. Dec. 19, 2019). In White, the special master found in favor of entitlement, however, this case can be distinguished based on the facts and expert opinions offered. The minor in White, A.W., received her third HPV vaccination on July 30, 2013, and 27 days later she had severe upper back pain, weakness, and difficulty walking. Id. at *25-26. She was subsequently diagnosed with TM. Id. at *25. Her clinical course was consistent with TM, her MRIs showed abnormalities also consistent with TM, and extensive testing for alternative causes, including EBV, were negative. Id. at *5, *25. Dr. Steinman was the expert for the petitioner in White and he noted that the minor, A.W., tested negative for any potential infectious agents, whereas here, Petitioner's testing was positive for EBV. Id. at *24-25. Further, in White, onset was 27 days post-vaccination. Id. at *26. Regarding Althen prong one, Dr. Steinman provided additional evidence and literature to support his theory in White, which was not provided in this matter. Id. at *21-24. Further, the special master in White found Dr. Steinman was "able to demonstrate there were sufficient homologies between the basic myelin protein and two of the HPV L1 stains (HPV6 and HPV 11) and between MOG and [] four HPV antigens in the vaccine." Id. at *24. Dr. Steinman did not opine that MOG was the host target here, and he did not propose there were four relevant HPV antigens. As such, White is distinguishable from Petitioner's case.

In summary, Petitioner has failed to offer a sound and reliable medical theory in support of her claim, and has thus failed to provide preponderant evidence to support Althen prong one.

2. Althen Prong Two

Under Althen prong two, Petitioner must prove by a preponderance of the evidence that there is a "logical sequence of cause and effect showing that the vaccination was the reason for the injury." Capizzano, 440 F.3d at 1324 (quoting Althen, 418 F.3d at 1278). "Petitioner must show that the vaccine was the 'but for' cause of the harm . . . or in other words, that the vaccine was the 'reason for the injury.'" Pafford, 451 F.3d at 1356 (internal citations omitted).

A petitioner need not make a specific type of evidentiary showing, i.e., "epidemiologic studies, rechallenge, the presence of pathological markers or genetic predisposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect." Capizzano, 440 F.3d at 1325. Instead, Petitioner may satisfy her burden by presenting circumstantial evidence and reliable medical opinions. Id. at 1325-26.

Since Petitioner failed to prove Althen prong one, it follows that she cannot prove Althen prong two. However, even if Petitioner had proven Althen prong one, the undersigned finds there is not preponderant evidence in the record to support a logical sequence of cause and effect showing her June 7, 2019 HPV vaccination to be the cause of her TM.

First, Petitioner's clinical course is not consistent with a vaccine-related condition. Petitioner received her third HPV vaccination on June 7, 2019. When she presented to the ED on June 10, she reported "[three] days of progressively worsening lower extremity weakness." Pet. Ex. 4 at 315. During her three-day hospitalization, she reported she felt "itchy and fatigue" and had malaise on June 7; had "generalized weakness[,] difficulty walking, [and] [two] falls" on June 8; and noticed "her legs felt heavy" and had "extremity weakness," occasional leg spasms, and falls on June 9. Id. at 311-12, 315, 319. Petitioner received three days of Solu-Medrol prior to discharge on June 13. At discharge, Petitioner was "walking well independently" and reported she "fe[lt] her strength [was] pretty much back to normal." Id. at 322. In 2019, Petitioner tested negative for autoimmune markers NMO, MOG, and AQP4, which are associated with neuroinflammatory disease.

This clinical course is not consistent with a vaccine-related condition for the reasons described above in Althen prong one and below in Althen prong three. To summarize, Petitioner's neurologic onset was one or two days post-vaccination, which is too short, even with a recall response, to be consistent with the mechanism of molecular mimicry.

Second, the undersigned finds that while some treating physicians noted a temporal association with Petitioner's recent HPV vaccination, none provided the basis for their opinion, or attributed Petitioner's TM to her HPV vaccination.

Dr. Ruschman wrote Petitioner "recently had her third HPV vaccine." Pet. Ex. 4 at 314. Dr. Opii documented "[Petitioner] mentioned that she received the third dose of [HPV] vaccine on Friday," June 7. Id. at 315. Neurologist Dr. Sheffield documented Petitioner's recent HPV vaccination in her history. Id. at 319. Assessment was "[m]yelopathy—likely [TM] with negative scan, [o]nset post vaccination." Id. at 321. Hospitalist Dr. Woody saw Petitioner on June 12, 2019 and she assessed Petitioner with "[TM] after HPV vaccination." Id. at 345. Hospitalist Dr. Furlow, at discharge, documented Petitioner presented with "worsening bilateral leg strength and difficulty walking after [a] recent HPV vaccination." Id. at 322. Each treating physician (Dr. Ruschman, Dr. Opii, Dr. Sheffield, Dr. Woody, and Dr. Furlow) merely provided recognition of a temporal relationship; none opined as to causation or attributed her vaccination to her TM. And as such, these statements are not persuasive evidence of vaccine causation. See Isaac v. Sec'y of Health & Hum. Servs., No. 08-601V, 2012 WL 3609993, at *26 (Fed. Cl. Spec. Mstr. July 30, 2012) ("A treating physician's recognition of a temporal relationship does not advance the analysis of causation.").

Generally, treating physician statements are typically "favored" as treating physicians "are likely to be in the best position to determine whether a 'logical sequence of cause and effect show[s] that the vaccination was the reason for the injury.'" Capizzano, 440 F.3d at 1326 (quoting Althen, 418 F.3d at 1280). However, no treating physician's views bind the special master, per se; rather, their views are carefully considered and evaluated. § 13(b)(1); Snyder, 88 Fed. Cl. at 746 n.67. "As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases." Welch v. Sec'y of Health & Hum. Servs., No. 18-494V, 2019 WL 3494360, at *8 (Fed. Cl. Spec. Mstr. July 2, 2019). An opinion by a treating physician that is not

supported by a factual basis or other evidence is conclusory in nature. See Robertson v. Sec'y of Health & Hum. Servs., No. 18-554V, 2022 WL 17484980, at *17 (Fed. Cl. Spec. Mstr. Dec. 7, 2022); Cedillo v. Sec'y of Health & Hum. Servs., 617 F.3d 1328, 1347 (Fed. Cir. 2010).

Following Petitioner's hospitalization, Dr. Furlow classified Petitioner's condition as "[a]cute [TM] possibly due to recent [HPV] vaccination" in an email to hospital administration on June 26, 2019. Pet. Ex. 4 at 531. And, on August 16, 2021, PA Hollingshead wrote a letter stating Petitioner "has a history of [TM] secondary to HPV vaccination." Pet. Ex. 29 at 294. The undersigned also finds these treating physician's statements are not persuasive.

First, Dr. Furlow used the word "possibly." An opinion expressed as a possibility is not sufficient to establish causation. See, e.g., Waterman, 123 Fed. Cl. at 573-74; Moberly, 592 F.3d at 1322 (emphasizing that "proof of a 'plausible' or 'possible' causal link between the vaccine and the injury" does not equate to proof of causation by a preponderance of the evidence).

Second, neither statement contains any explanation in support of vaccine causation. An opinion by a treating physician that is not supported by a factual basis or other evidence is conclusory in nature. See Robertson, 2022 WL 17484980, at *17; Cedillo, 617 F.3d at 1347. And special masters consistently reject conclusory expert statements. Kreizenbeck, 2018 WL 3679843, at *31.

Lastly, the undersigned finds PA Hollingsworth's letter is not persuasive because (1) it does not appear that PA Hollingsworth treated Petitioner at or around the time of injury, unlike Dr. Furlow, Dr. Ruschman, Dr. Opii, Dr. Sheffield, and Dr. Woody; and (2) this letter was provided two years after vaccination and injury and for the purpose of obtaining an exemption from the Covid-19 vaccination. See Zumwalt v. Sec'y of Health & Hum. Servs., No. 16- 994V, 2019 WL 1953739, at *19 (Fed. Cl. Spec. Mstr. Mar. 21, 2019) (rejecting opinion from a treating provider when he presented an opinion two-and-one-half years after treatment and after litigation was initiated), mot. for rev. den'd, 146 Fed. Cl. 525 (2019).

Lastly, Respondent's expert Dr. Matloubian opined that there were other causes for Petitioner's complaints, including EBV. Dr. Steinman opined "[e]xtensive studies for infectious, neoplastic, and structural causes for TM failed to provide a satisfactory alternative cause." Pet. Ex. 11 at 5. However, Dr. Steinman did not refute Dr. Matloubian's opinion. Dr. Steinman did not address whether Petitioner's EBV results affected his opinion.

The undersigned acknowledges that Petitioner is not required to eliminate other potential causes in order to be entitled to compensation. See Walther v. Sec'y of Health & Hum. Servs., 485 F.3d 1146, 1149-52 (Fed. Cir. 2007) (finding a petitioner does not bear the burden of eliminating alternative independent potential causes). However, she finds it reasonable to consider "evidence of other possible sources of injury" to determine "whether prima facie showing has been made that the vaccine was a substantial factor in causing the injury in question." Stone, 676 F.3d at 1379; see also Winkler v. Sec'y of Health & Hum. Servs., 88 F.4th 958, 963 (Fed. Cir. 2023) (finding that the special master's "contemplation of a potential causative agent when evaluating whether or not a petitioner has established a prima facie case is in accordance with the law").

For the reasons described above, the undersigned finds Petitioner has not proven by preponderant evidence a logical sequence of cause and effect establishing that her HPV vaccination caused her to develop TM. Thus, Petitioner has not satisfied the second Althen prong.

3. Althen Prong Three

Althen prong three requires Petitioner to establish a "proximate temporal relationship" between the vaccination and the injury alleged. Althen, 418 F.3d at 1281. That phrase has been defined as a "medically acceptable temporal relationship." Id. A petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." de Bazan, 539 F.3d at 1352. The explanation for what is a medically acceptable time frame must also coincide with the theory of how the relevant vaccine can cause the injury alleged (under Althen prong one). Id.; Koehn v. Sec'y of Health & Hum. Servs., 773 F.3d 1579, 1243 (Fed. Cir. 2014); Shapiro, 101 Fed. Cl. at 542.

Before addressing Althen prong three, it is appropriate to first determine date of onset. The undersigned finds onset to be on June 8 or June 9, 2019, one or two days after vaccination.

Petitioner received her HPV vaccination on June 7, 2019. Petitioner's expert, Dr. Steinman opined Petitioner's onset of TM was within one to two days after her third HPV vaccination. Respondent's expert, Dr. Jamieson opined Petitioner had fatigue and leg weakness on the day of vaccination, June 7, 2019. Dr. Matloubian agreed Petitioner's onset was "rapid," but he did not specifically provide an opinion as to the date of onset.

An examination of the medical records shows three treating physicians documented reports from Petitioner that neurologic symptoms (leg weakness, difficulty walking, and falls) began on June 8 or June 9. Dr. Ruschman, at the ED on June 10, wrote Petitioner reported feeling "itchy and fatigue" shortly after vaccination on June 7, which worsened the following day (June 8), and led to "extremity weakness," occasional leg spasms, and falls on June 9. Pet. Ex. 4 at 311-12.

Dr. Opii, who saw Petitioner shortly thereafter on June 10, documented Petitioner presented to the ED for "[three] days of progressively worsening lower extremity weakness." Pet. Ex. 4 at 315. This suggests onset began June 7, the day of vaccination. Petitioner reported "generalized weakness[,] difficulty walking, [and] [two] falls" on June 8 (one day post-vaccination). Id. She also reported the same symptoms on June 9 (two days post-vaccination).

Neurologist Dr. Sheffield examined Petitioner on June 11 and documented Petitioner noticed "her legs felt heavy," followed by multiple falls due to progressive weakness on June 9. Pet. Ex. 4 at 319.

Consistent with Petitioner's expert Dr Steinman as well as Petitioner's treating physicians (Dr. Ruschman, Dr. Opii, and Dr. Sheffield), the undersigned finds Petitioner's neurologic symptom onset was June 8 or June 9, 2019, one or two days post-HPV vaccination.

Dr. Steinman opines this timing is medically acceptable because (1) Petitioner had a recall response due to her two previous HPV vaccinations and (2) this timing is consistent with cases of GBS post-flu vaccination and other Vaccine Program cases in which Dr. Steinman has provided his expert opinion.[72]

First, with regard to Dr. Steinman's assertion that an onset of one to two days is appropriate with a recall response, the undersigned does not find his opinions persuasive, sufficiently explained, or consistent with the literature. Dr. Steinman did not sufficiently explain a "recall response" or how it could account for an onset of one day or two days post-HPV vaccination.

The IOM discusses the latency period[73] between antigen exposure and peak adaptive immune response. This literature explains that with subsequent exposures—i.e., multiple HPV vaccinations—the latency period is shorter, with a lag phase[74] of one to three days followed by a

[72] Again, special masters' decisions are not binding on the undersigned. See Hanlon, 40 Fed. Cl. at 630 (1998); Boatmon, 941 F.3d at 1358. Of note, an onset of one day was not found to be medically appropriate in a case of TM in which Dr. Steinman posited a similar theory and explanation as to timing. See, e.g., Greenslade, 2024 WL 3527665, at *37-41 ("[P]etitioner has not preponderantly shown that a 24-hour onset of TM is 'medically acceptable' even following a recall response.").

[73] A latent period is "a seemingly inactive period, such as that between exposure to an infection and manifestation of symptoms (incubation p[eriod]) or between the presentation of a stimulus and the response (latency [])." Latent Period, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=97284 (last visited Mar. 18, 2025). Latency refers to "the time between the instant of stimulation and the beginning of a response." Latency, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=27698 (last visited Mar. 18, 2025). The IOM explained "the latency between the first (primary) exposure and development of the primary response is characterized by a lag phase, logarithmic phase, and plateau phase." Pet. Ex. 28 at 87. "[T]he latency between subsequent exposure to the antigen and development of the immune response will usually be shorter. The lag phase is generally [one] to [three] days; the logarithmic phase of the secondary antibody response occurs over the next [three] to [five] days." Id.

[74] A lag phase is characterized by "activation of B and T cells upon encounter with the antigen for which they are specific, and this triggers the cells' differentiation into effector and memory cells." Pet. Ex. 28 at 87.

logarithmic phase[75] of three to five days, resulting in a latency period ranging from four to eight days.

A latency period "is characterized by a lag phase, logarithmic phase, and plateau phase." Pet. Ex. 28 at 86-87. Petitioner and Dr. Steinman failed to explain how this would allow for an onset of one to two days. Dr. Steinman did not explain how a shortened lag phase, reflecting activation of B and T cells upon an encounter with the antigen for which they were specific, would trigger the initial manifestation of symptoms Petitioner experienced on June 8 or 9, 2019. In other words, Dr. Steinman failed to account for all of the phases of the latency period, that period between vaccination and manifestation of symptoms, or explain how they could occur in one to two days, even considering Petitioner's prior exposure to the antigen.

Additionally, Petitioner's one to two day onset of symptoms is not consistent with the literature. Petitioner does not provide any literature specific to TM post-HPV vaccination. And, none of the literature provided by Petitioner supports a one to two day onset of TM.

Dr. Steinman cites to Lai et al., arguing this study provides support that a recall response can occur within six hours. However, Dr. Steinman does not explain this study and how it specifically applies to TM, HPV vaccination, and the facts and circumstances of this present case. The undersigned thus does not find this reference to be persuasive evidence to support Althen prong three.

Dr. Steinman also attempts to rely on GBS data to support his opinion that such a rapid onset is medically acceptable. He cited to Schonberger et al., who found cases of GBS occurred between day zero (date of vaccination) and day one. However, the Schonberger et al. article was not filed. Based on the undersigned's knowledge of this study, Schonberger et al. did not explain or discuss TM cases and thus, the data is not applicable in the context of TM, which affects the spinal cord and is a different illness than GBS, which is a PNS demyelinating illness. Moreover, the onset period for GBS following flu vaccination as set forth in the Vaccine Injury Table is three to 42 days. 42 C.F.R. § 100.3(a)(XIV)(D). Thus, even if the undersigned used GBS data for this TM case, the timing would be inconsistent.

Lastly, the undersigned notes decisions from other special masters where petitions alleging vaccine-induced TM in the Program have been dismissed for similar onset found too close in time to vaccination to be medically reasonable. See, e.g., Martinez ex rel. W.M. v. Sec'y of Health & Hum. Servs., No. 16-738V, 2022 WL 4884923, at *27 (Fed Cl. Spec. Mstr. Sept. 9, 2022) ("Because TM is reasonably understood to be mediated by an autoimmune reaction involving antibodies or other immune cells associated with the adaptive, lagging immune response in reaction to antigenic exposures . . . , a relatively short onset timeframe is simply not medically acceptable." (emphasis omitted)); Palattao v. Sec'y of Health & Hum. Servs., No. 13-591V, 2019 WL 989380, *35 (Fed. Cl. Spec. Mstr. Feb. 4, 2019) (citing dismissals of cases with 24-hour onset for vaccine-related TM and finding a 30- to 36-hour onset to not be medically acceptable); Brancheau v. Sec'y of Health & Hum. Servs., No. 21-1209V,

[75] "For B cells, the logarithmic phase is characterized by an increase in serum antibody levels that classically is logarithmic." Pet. Ex. 28 at 87.

2024 WL 1619606, at *23-26 (Fed. Cl. Spec. Mstr. Mar. 21, 2024) (finding a one day onset of TM following flu vaccination not appropriate given the theory of molecular mimicry); Forrest v. Sec'y of Health & Hum. Servs., No. 14-1046V, 2019 WL 925495, at *6, *8 (Fed. Cl. Spec. Mstr. Jan. 28, 2019) (finding "a preponderance of the evidence shows that molecular mimicry is not likely to happen within 36 hours, even for a recall response"); Mosley v. Sec'y of Health & Hum. Servs., No. 08-724V, 2015 WL 2354316, at *19 (Fed. Cl. Spec. Mstr. Apr. 27, 2015) (denying compensation where "onset of TM one day after tetanus vaccination [was] too soon to support vaccine causation"); Jagoe v. Sec'y of Health & Hum. Servs., No. 08-678V, 2012 WL 13036265, at *28 (Fed. Cl. Spec. Mstr. Aug. 3, 2012) (finding a 24-hour onset not medically appropriate for a vaccine-induced TM injury); Crosby v. Sec'y of Health & Hum, Servs., No. 08-799V, 2012 WL 13036266, at *38-39 (Fed. Cl. Spec. Mstr. June 20, 2012) (same).

While the above cases are not binding here, the undersigned agrees with the reasoning of other special masters as it relates to onset and Althen prong three. See Boatmon, 941 F.3d at 1358; Hanlon, 40 Fed. Cl. at 630.

In Forrest, for example, the petitioner's expert proposed that the flu vaccine can cause TM via molecular mimicry. 2019 WL 925495, at *3. To explain the 36-hour onset, the expert proposed the Forrest Petitioner had a recall response due to previous flu vaccinations. Id. at *4. The special master found that "[e]ven if molecular mimicry could be accepted to explain how the flu vaccine can cause [TM] abstractly, . . . a preponderance of the evidence shows that molecular mimicry is not likely to happen within 36 hours, even for a recall response." Id. at *6. The special master similarly discussed the IOM report that referenced the immune response latency period to include a lag phase and logarithmic phase. Id. Petitioner's expert in Forrest, like Dr. Steinman, "cited the IOM for the basis that the lag phase generally can be as short as one day," but failed to persuasively address the logarithmic phase and the timing and presentation of symptoms associated with the logarithmic phase.[76] Id.

In summary, the undersigned finds (1) by preponderant evidence that the onset of Petitioner's TM was June 8 or June 9, 2019, one to two days day after her third HPV vaccination, and (2) there is a lack of preponderant evidence to show that an onset of one to two days is appropriate given the proposed mechanism of molecular mimicry.

Therefore, the undersigned finds the temporal association is not appropriate given the mechanism of injury. Petitioner has failed to satisfy the third Althen prong.

V. CONCLUSION

The undersigned extends her sympathy for the suffering Petitioner has experienced due to her illness. However, the undersigned's Decision cannot be based on her sympathy, but must be based on the evidence and the law.

[76] Forrest also provides a more thorough explanation of the latency period of the immune response and the lag and logarithmic phases. See Forrest, 2019 WL 925495, at *6-7. In the present case, these concepts were not discussed by Dr. Steinman.

For the reasons discussed above, the undersigned finds that Petitioner has failed to provide preponderant evidence of causation, and therefore, the petition must be dismissed.

IT IS SO ORDERED.

s/Nora Beth Dorsey
Nora Beth Dorsey
Special Master